06355

U. S. NAVAL AIR DEVELOPMENT CENTER

Johnsville, Warminster, Pennsylvania

NADC-MR-6305 1 August 1963

Effect of Physical and Psychic Stress
On Phospholipid Fatty Acid and Related Phosphorus Lipids

Bureau of Medicine and Surgery

DEPARTMENT OF THE NAVY
U. S. NAVAL AIR DEVELOPMENT CENTER
JOHNSVILLE
WARMINSTER, PA. 18974

Aerospace Medical Research Department

NADC-MR-6805 1 August 1968

Effect Of Physical And Psychic Stress On Phosphatidyl Glycerol And Related Phospholipids

Naval Air Systems Command
Air Task A34 531 062/2021/F022 01 06
(5314-062-59)
Air Task R01 101 01 (RC-3-01)

Bureau of Medicine and Surgery
Task MR005.04.0098
Task MF12.524.005-7006

Prepared by:

B. David Polis, Ph.D

Edith Polis, M.S.

John deCani, Ph.D.

H. P. Schwarz, M.D.*

Lorraine Dreisbach, M.S.*

Approved by:

Carl F. Schmidt, M.D.
Research Director
Aerospace Medical Research Department

Released by:

D. P. Morris, CAPT, MC, USN
Director
Aerospace Medical Research Department

*Department of Biochemistry, Philadelphia General Hospital, Philadelphia, Pa.

ACKNOWLEDGMENT

The technical assistance of Miss Rosalie Bernstein and Mrs. Joan Turnoff and the secretarial work of Mrs. Carole Marks are gratefully acknowledged.

ADMINISTRATIVE INFORMATION

Supported in part by AEC Contract AT(13-1)1864.

SUMMARY

Chromatographic analyses of phospholipids in tissue and plasma of rats exposed to lethal levels of ionizing radiation or acceleration stress yielded a consistent pattern of increased concentrations of phosphatidyl glycerol. Extension of the studies to humans stressed by acceleration to grayout, sleep deprivation, schizophrenia, combat, etc., revealed that all stresses were accompanied by significant increments in plasma phosphatidyl glycerol. Moreover, the stressed populations could be distinguished from each other when the changes in phosphatidyl glycerol were related to concomitant variations in seven other phospholipids. Using a statistical method involving discriminant function analysis, a function Z was obtained which represented the summation of the log of each individual phospholipid concentration times a distributive constant. With this analysis the phospholipid distribution separated normal, combat, and schizophrenic populations into three distinct groups.

In animal experiments, it was found that hypophysectomy, which markedly enhanced the tolerance of the rat to acceleration stress, abolished the plasma changes of acceleration and caused a two-fold increase in the brain level of phosphatidyl glycerol after exposure to acceleration. The results from human and animal experiments are interpreted to indicate that some center of the brain can interpret certain sensory inputs as threats to survival and reacts by mobilizing biochemical factors at a molecular level to meet this threat and enhance survival. In all the stresses studied, with the exception of schizophrenia, the individuals returned to normal levels with rest. The maintenance of this "locked stress" pattern in the schizophrenic and the reversible plasma phospholipid changes in the other physical and psychic stresses in human, coupled with the tissue phospholipid changes in rat, offer an approach to the cerebral control factors operative in the biochemistry of environmental and pathological stress.

TABLE OF CONTENTS

LIST OF FIGURES

LIST OF FIGURES (CONT'D)

LIST OF TABLES

INTRODUCTION

Acting on the precept that answers to the problems generated by environmental and pathological stress might be found in the mechanisms of biological energy control and utilization, and predicating the approach on the premise that common bioenergetic parameters were operative in stresses of diverse etiology, a molecular probe was sought to reveal pertinent energetic and regulatory pathways.

Experiments with isolated particulate enzyme systems concerned with oxidative metabolism and cellular energy utilization revealed unusually fast turnover rates in the membranous lipid-containing sub-structures involved in the transfer of "group potential energy" (1). The identification of one of these molecules as phosphatidyl glycerol was accompanied by the finding of a marked increase in content and a decrease in turnover of this phospholipid in the first twenty-four hours after exposure to lethal levels of ionizing radiation (1).

Following the radiation study, the experimental work was extended to another physiological stress to determine whether the changes observed were specific for ionizing radiation or were the result of a more general molecular response to stress. To contrast with the relatively long-term effects of ionizing radiation, a short anoxic and fatigue stress like acceleration was chosen. Since hypophysectomy markedly enhanced the tolerance of the rat to acceleration (2), the effect of this surgical procedure on the phospholipid pattern before and after acceleration also was investigated. In addition, the phospholipid analyses which previously were limited to liver were extended to include brain and plasma.

With the phospholipid changes in tissue and plasma of the stressed rat as a prologue, attention was directed to the changes that occurred in stressed humans. For obvious reasons plasma was the sole tissue investigated. The effect of physical stress on plasma phospholipids in humans was first investigated in young Navy personnel exposed to acceleration. The indications of psychic factors operating during the control period prior to acceleration led to an investigation of extreme psychic stress. For this study hospitalized chronic schizophrenic patients were used. As an example of the effects of fatigue, the study was extended to sleep-deprived subjects. Following these studies, the effect of combat stress, which represented a complex interaction of physical, fatigue, and psychic factors, was investigated. Animal experimentation suggested that cerebral control factors may be involved in the plasma phospholipid patterns. Therefore, a group of children with recognized brain injury resulting in mental retardation was investigated and compared to a similar age group of mentally normal but hospitalized convalescent children.

METHODOLOGY

A. Chemical

The analyses of the individual phospholipids were carried out essentially by the method of Dawson et al. (3). The blood samples were centrifuged immediately and the plasma frozen and stored or, when possible, extracted immediately in 20 volumes of chloroform-methanol (2:1 v/v). No significant changes in the phospholipids analyzed were observed during the freezing and storage. Tissues were weighed and homogenized in 50 volumes of chloroform-methanol (2:1 v/v) per gram wet weight of tissue. Under these conditions the lipids were reproducibly extracted from the homogenates within 24 to 48 hours at room temperature in the dark. The protein precipitate was filtered off on sintered glass crucibles and dried in-vacuo to constant weight at room temperature. The filtrate was evaporated to dryness in-vacuo at 35°C and then dissolved in chloroform-methanol-water (60:30:4.5 v/v) using 5 ml of solvent per gram wet weight of original protein. This solution was passed through a 6 mm inner diameter column containing 2 grams Sephadex-G-25 for the removal of non-lipid water soluble phosphate compounds (4) and then was eluted with 8 ml of chloroform-methanol (2:1 v/v). The total eluate was evaporated to dryness under a stream of nitrogen at 30°C and the residue dissolved in 25 ml of chloroform. After an aliquot was taken for the determination of total lipid phosphorus, the chloroform solution of phospholipids was applied to a column containing 5 g silicic acid and the column was washed with 150 ml of chloroform to remove non-phospholipid material like cholesterol, cholesterol esters, free fatty acids, and tri-glycerides. The adsorbed phospholipids were eluted with methanol (150 ml). An aliquot of the phospholipid extract containing about 100-500 µg of lipid phosphorus was evaporated to dryness in a nitrogen stream and subjected to mild alkaline hydrolysis with .03 N NaOH in methanol for 20 minutes at 37°C. The hydrolysate was neutralized with an amount of ethylformate equivalent to the NaOH and again evaporated to dryness in a nitrogen stream. The residue was distributed between one volume of water and two volumes isobutanol-chloroform (1:2 v/v) and the two phases separated by centrifugation. An aliquot of the deacylated "alkali labile" phospholipids contained in the water phase was spotted on Whatman #3 paper and the glycerol phosphate components separated first by descending chromatography in phenol saturated with water-acetic acid-ethanol (50:5:6 v/v). After development for 16 hours, the solvents were removed and resolution in the second dimension was accomplished by high voltage ionophoresis in a buffer solution of pyridine-acetic acid-water (1:10:89 v/v) at pH 3.6 using a current of 100 to 125 ma at 2000 V for 1 1/2 hours. The chromatograms were sprayed with ninhydrin reagent to locate amino lipids and afterwards with acid molybdate (5) to determine the phosphorus compounds. Choline-containing compounds were detected with Dragendorff's reagent when necessary. Analyses of the alkali stable phospholipids (plasmalogen, sphingomyelin, alkyl ethers) contained in the chloroform

layer of the phase distribution of the hydrolysates were carried out exactly as described by Dawson et al. (3). The individual spots obtained by the two-dimensional chromatography were identified from the Rfs found with deacylated purified standards. Quantitation of the individual phospholipids was accomplished by digestion of the stained spots with 72% v/v perchloric acid and subsequent determination of the inorganic phosphorus (6).

In the course of the chromatographic analysis of phospholipids by Dawson's procedure, it was found by Schwartz et al. (1) that, although most of the phosphatidyl glycerol was deacylated to α-glycerylphosphoryl glycerol, a small fraction was hydrolyzed to an isomer of α-glycerylphosphoryl glycerol which did not separate with the techniques used from α-glycerophosphate derived from phosphatidic acid. This was made evident by the glycerol-to-phosphorus ratio of 1.88 to 1.0 instead of 1 to 1 that would have been expected from phosphatidic acid. In the data described, phosphatidic acid therefore is not pure per se and represents a mixture of phosphatidic acid and an isomeric form of phosphatidyl glycerol. Since no distinction can be made between the level of this isomer that occurs naturally and that which may be formed by the deacylation procedure, no adequate corrections can be applied. This fraction is then labelled as "Phosphatidic Acid" because it migrates with α-glycerophosphate, even though a major fraction of this component is phosphatidyl glycerol isomer. Approximately 6% of the large lecithin fraction and 40% of the phosphatidyl inositide are converted to cyclic glycerophosphoric acid during hydrolysis (3). Adequate corrections for these changes were made.

B. Stress Procedures

1. Animal Studies

X-ray irradiation experiments were carried out as described previously (1). Female albino Wistar rats, weighing approximately 200 grams, were kept on a balanced diet *ad libitum*. Food was withdrawn the night before the experiment and groups of three rats were placed in thin-walled plastic containers and exposed to a single 3000 r whole body dose of X-rays from a Willard-Westinghouse machine at 200 kw and 20 mA, using 1 mm aluminum and 0.5 mm copper filters. Twenty-four hours later blood was drawn from the hearts of the irradiated and control rats and the livers were removed for analysis.

Phospholipid changes in acceleration stress were evaluated in Sprague-Dawley rats (250 g) accelerated at 20 G on an eight-foot centrifuge. Each animal was restrained in a wire cage mounted with the long axis of the body parallel to the radius of the centrifuge and the head facing the center (positive or $+G_z$ acceleration). The endpoint of tolerance to the acceleration stress was monitored by the ECG obtained with suture clip electrodes inserted in the skin at the chest and back and recorded through transistorized amplifiers (2, 7). At the termination of the run, the animal was killed by cervical dislocation, blood was obtained by cardiac puncture and the

brain and liver were removed and homogenized in chloroform-methanol (2:1 v/v). For controls, the animals were restrained and subjected to sham runs for time periods equivalent to the duration of the acceleration runs. These showed insignificant changes in phospholipid distribution when compared to unrestrained normal animal controls.

Pilot studies on the sampling procedures showed that the lowest consistent values of phospholipids in plasma were obtained when the animals were killed by cervical dislocation followed immediately by heart puncture. Even though the sampling technique may have introduced some element of uncertainty about the normal levels of the phospholipids analyzed, the differences between control and stressed animals are those superimposed on the manipulative stress of sampling and may be considered in this light with the stated reservations.

2. Human Studies

(a) Acceleration Stress

Acceleration experiments were carried out on the 50-foot radius dynamic flight simulator of the Naval Air Development Center at Warminster, Pennsylvania. This unique centrifuge for humans and its computer control system were operated by the Dynamic Simulation Division headed by R. Crosbie (8) and associates, P. Edwards and M. Freed. Navy volunteers, 20-35 years of age, were exposed to acceleration in the G_z position (head-to-foot) for 10-second intervals starting at 3.0 G and increasing by 0.5 G increments for successive 10-second runs to a maximum of 5.0 G, during which time the subjects suffered peripheral light loss and signaled the experience of grayout or blackout. At this point the individual experiment was terminated. Blood samples were drawn from the cubital vein before and immediately after the acceleration.

Subsequently, a time study of the phospholipid levels in blood during the 24 hours following the acceleration procedure was carried out. In this study, blood samples were drawn before acceleration, immediately after acceleration, three hours and twenty-four hours later. These experiments were complemented by a similar study of the diurnal variation in phospholipid levels during the course of an average work day. Here, samples were drawn before breakfast (8:00 a.m.) after breakfast (9:30 a.m.), after lunch (1:00 p.m.), and at the end of the work day (3:30 p.m.). All these studies were conducted under the medical supervision of Drs. R. Patton and R. Oleynik of the U.S. Navy after a preliminary study on the biochemical effects of acceleration stress on four subjects was carried out with Dr. E. York, M.C., U.S.N. (9).

(b) Schizophrenia

As an example of extreme psychic stress, 20 patients from the New Jersey Neuropsychiatric Institute were studied. These subjects, designated as

long-term chronic schizophrenics, so called "back-ward" patients, were selected by Dr. Joseph Noval of that institute. The blood plasma samples were obtained between 8 and 9 a.m. before breakfast. These patients were part of a study on the effects of various drugs on schizophrenia. The blood samples for phospholipid analysis were collected during a "washout" period of 4 weeks between the different drug trials. Cortisol and corticosterone analyses were determined by Drs. J. Noval and T. Post on the plasma from the schizophrenic and other stress studies. These findings will be reported by them elsewhere.

(c) Sleep Deprivation

For the condition of mental and physical fatigue, blood samples were obtained from a study of sleep deprivation on 7 male Navy personnel, 25-35 years old, conducted by Drs. R. Squires and R. Oleynik who were investigating electroencephalographic changes induced by this stress. This work and possible correlations between the electroencephalographic changes and the biochemical data will be reported separately by them; here we detail the plasma biochemical changes resulting from the stress. In this study, blood samples were collected at 3 p.m. of a control day starting at 6 a.m. After sleep deprivation for 33 hours the blood was sampled again at 3 p.m. A recovery blood specimen was obtained after 24 or 48 hours of rest, again at 3 p.m. There were no dietary restrictions during this study.

(d) Combat Stress

The blood samples from military personnel in combat were supplied by Captain Frank H. Austin, Jr., M.C., U.S.N., who headed a multidisciplinary group from Navy, Air Force, and NASA to investigate psychophysiological factors relating to acute and cumulative fatigue of naval aviators. Blood samples were obtained from aviators from an attack aircraft carrier operating against heavily defended targets in North Viet Nam. This occurred near the end of the Air Wing's second combat cruise and the termination of a 7-month deployment. The sampling was from 27 aviators during the 86th through the 108th day of line combat operation. Since sampling was necessarily made on a volunteer and non-interference basis, there were no restrictions on diet and the time of collection varied throughout the day. All bloods were drawn after carrier landing and debriefing from the combat mission. Complete details of this psychophysiological study are reported elsewhere (10).

(e) Mentally Retarded and Convalescent Children

For comparison with the results of the previous studies, a group of mentally retarded children with "permanent brain damage" produced, presumptively, by episodes of anoxia during birth, is included. These children, varying in age from 1 1/2 to 17 years, were selected by Dr. L. Green.

Since the age group differed significantly from the normal series, an additional control group of convalescent children was obtained by Dr. C. Kennedy. These ranged in age from 10 months to 15 years.

RESULTS

A. Correlation of Tissue and Plasma Changes in X-irradiated and Acceleration Stressed Rats

Of the 19 phospholipid species that were analyzed in blood plasma, liver, and brain of control and stressed rats, the dominant and most interesting changes were observed with phosphatidyl glycerol. The data listed in Table I emphasizes the similarities in content and direction of phosphatidyl glycerol change induced, both in plasma and tissue, by two unrelated stresses. For purposes of comparison, the concentration levels of phosphatidyl glycerol are listed as the per cent of the total phospholipid. The total phospholipids are listed in μM of lipid phosphorus per liter of plasma or per gram of dry weight of tissue. It may be emphasized that while the changes in total phospholipids are in the order of 10-30 per cent and, with the exception of the variation caused by X-ray, are not significant, the stress-induced changes in phosphatidyl glycerol are in the order of 100-300 per cent and are markedly significant.

In agreement with the data presented by Entenman et al. (11), X-ray irradiation caused a significant elevation of total lipid phosphorus of blood plasma but no significant change in liver. Acceleration stress on the other hand caused no significant increase in the total phospholipid of the plasma, brain, or liver. However, a significant increase in the levels of phosphatidyl glycerol took place in plasma and liver after acceleration and after X-irradiation. Thus, a striking similarity of effects is found in plasma and liver, irrespective of the nature of the stress.

B. Pituitary Control of Plasma Phosphatidyl Glycerol Levels of Acceleration Stressed Rats

The procedure of hypophysectomy per se causes no significant changes in the plasma phospholipid pattern of unstressed rats. Some exceptional effects, however, were found in the phosphatidyl glycerol changes of the hypophysectomized rat after acceleration. These effects were:

1. The phosphatidyl glycerol changes induced by acceleration stress in the plasma of normal rats did not occur.

2. A significant increase in phosphatidyl glycerol content of the hypophysectomized rat brain did occur.

The stress induced variations in liver remained unaffected by hypophysectomy.

Recalling that hypophysectomy increased the tolerance of rats to acceleration stress (2) to the degree that they survived for a mean time of 40 minutes at 20 G compared to 9 minutes for the normal controls, then the

TABLE I

THE EFFECT OF ACCELERATION OR IRRADIATION
ON PLASMA AND TISSUE PHOSPHOLIPID LEVELS OF NORMAL AND HYPOPHYSECTOMIZED RATS

Phospholipid		ACCELERATION				IRRADIATION WITH 3000 r	
		Normal Rat		Hypophysectomized Rat		Normal Rat	
		Control	Acceleration	Control	Acceleration	Control	Irradiation
Phosphatidyl Glycerol		Per Cent of Total Phospholipid					
	Plasma	1.0 ± .1 (27)	2.3** ± .3 (21)	1.0 ± .3 (32)	1.0 ± .1 (45)	1.0 ± .1 (24)	1.8** ± .1 (24)
	Liver	1.0 ± .1 (8)	2.2** ± .2 (6)	1.3 ± .1 (6)	2.0** ± .1 (7)	1.6 ± .1 (33)	2.1* ± .1 (60)
	Brain	1.5 ± .1 (10)	2.5 ± 1.2 (11)	1.9 ± .3 (15)	3.4* ± .4 (22)	—	—
Total Lipid Phosphorus		µM lipid P/liter plasma					
	Plasma	1921.6 ±91.0 (27)	1633.4±196.5 (21)	1597.0±345.7 (32)	1464.7±293.6 (45)	1497.5±113.5 (24)	1998.9*±135.8 (24)
		µM lipid P/gram dry residue					
	Liver	120.5 ± 6.3 (8)	144.0 ± 9.4 (6)	129.0 ± 3.8 (6)	142.1 ± 6.1 (7)	127.8 ± 5.4 (33)	137.8 ± 3.5 (60)
	Brain	491.4 ±12.6 (10)	520.5 ±25.6 (11)	562.3 ±16.4 (15)	601.8 ±34.7 (22)	—	—

Data represents mean plus or minus standard error for each group of rats.
( ) Number of rats
* P(t) = 1%
** P(t) = 0.1%

failure to find an increase in plasma phosphatidyl glycerol in accelerated hypophysectomized rats might be attributed to an increased utilization of this phospholipid during the prolonged acceleration stress. Alternatively, since the brain content of this phospholipid increased after acceleration only in hypophysectomized animals, while the liver content of phosphatidyl glycerol became elevated in both normal and hypophysectomized rats, it might be inferred that the plasma level increments of phosphatidyl glycerol in stressed unoperated rats came from the brain and was influenced by the pituitary. Irrespective of the validity of either hypothesis, it is apparent that the pituitary is involved in the plasma concentration of phosphatidyl glycerol and that directly or indirectly the molecule reflects the action of regulatory factors intimately involved in the tolerance to stress.

C. Effect of Stress on Human Plasma Phospholipids

The effect of acceleration stress on plasma phospholipids of 24 subjects compared to 32 normal controls are shown in Table II (See Appendix A for full data). While no significant change was observed in the total phospholipid levels, marked increments in the levels of phosphatidyl glycerol, "phosphatidic acid," and phosphatidyl ethanolamine were found that differed significantly from the control at the 1 per cent level of confidence or less. A decrement in cardiolipin, significant at the 5 per cent level of confidence, also was observed. Of the five phospholipid components changing significantly after acceleration the most dramatic increase occurred with phosphatidyl glycerol. In view of the comparable tissue and plasma changes of this compound found in accelerated rats, a more detailed investigation of factors affecting this phospholipid in humans was undertaken.

In Figure 1, the bar graphs show the increases in phosphatidyl glycerol that occur in each subject exposed to the acceleration stress. In every subject there is a significant rise in the level of phosphatidyl glycerol. It is apparent that the control levels of phosphatidyl glycerol vary considerably between individual subjects. In general, it was found that those subjects who reported anxiety over the proposed experiment or over unrelated emotional problems, usually ran levels of phosphatidyl glycerol which were significantly above the average range. Nonetheless, acceleration superimposed increments over and above these initial levels.

Any attempt to correlate variations in the concentration of individual phospholipids with etiological factors like stress is complicated by the undefined origin of the changes. These may be the result of the action of factors specific for the individual phospholipids or may represent a nonspecific change reflecting primarily a variation in the total phospholipid level. This uncertainty becomes especially pertinent in the comparison of human populations, uncontrolled with respect to nutritional variables, as well as other unrecognized factors. Some discrimination of the controlling mechanisms may be achieved by comparing the individual phospholipid changes as a per cent of the total phospholipid, as well as on an absolute concentration basis. Thus, a rise in total phospholipids with no change in the

TABLE II

STRESS INDUCED CHANGES IN HUMAN PLASMA PHOSPHOLIPIDS

Number of Subjects	32	24	20	7	16	15	9
Stress State	Normal	Acceleration	Schizophrenia	Sleep Deprivation	Extended Combat	Retarded Children	Convalescent Children
Phospholipid	μM P/liter plasma						
Total Lipid Phosphorus	2272 ±83.7	2271 ±91.1	*2621 ±79.4	2131 ±154.7	2936 ±128.8	2606 ±97.0	2501 ±194.8
Phosphatidyl Choline	1564 ±67.4	1507 ±68.7	1744 ±82.8	1401 ±124.1	*1908 ±100.9	1622 ±72.0	1647 ±127.6
Phosphatidyl Ethanolamine	41 ± 2.5	*54 ± 3.7	83 ± 6.6	47 ± 8.7	85 ± 7.3	*50 ± 2.9	36 ± 3.0
Phosphatidyl Serine	17 ± 1.4	19 ± 1.9	31 ± 2.7	23 ± 2.1	34 ± 4.1	22 ± 2.4	18 ± 1.7
Cardiolipin	30 ± 1.3	25 ± 2.3	40 ± 3.5	*25 ± 1.5	39 ± 3.4	30 ± 2.4	25 ± 3.3
Phosphatidic Acid	14 ± 0.9	*18 ± 1.3	36 ± 3.7	35 ± 2.0	27 ± 1.9	17 ± 2.2	14 ± 1.5
Phosphatidyl Glycerol	25 ± 2.0	53 ± 3.5	57 ± 3.9	41 ± 3.0	63 ± 4.8	44 ± 2.7	24 ± 2.9
Phosphatidyl Inositide	90 ± 7.3	83 ± 6.5	118 ±13.6	88 ± 10.3	95 ± 8.4	96 ± 8.4	82 ± 10.1
Cyclic Glycerophosphoric Acid	85 ± 8.1	85 ± 7.9	119 ±15.7	66 ± 6.2	103 ± 8.1	74 ± 4.8	84 ± 7.5
Inorganic Phosphate	12 ± 1.3	13 ± 1.3	20 ± 2.9	20 ± 3.5	20 ± 3.0	18 ± 2.6	17 ± 1.6
Choline Plasmalogen	41 ± 1.6	42 ± 1.9	55 ± 5.3	53 ± 11.8	59 ± 6.0	114 ±10.0	38 ± 2.4
Ethanolamine Plasmalogen	17 ± 1.6	17 ± 1.4	25 ± 3.1	23 ± 4.8	32 ± 5.1	18 ± 3.0	15 ± 1.7
Serine Plasmalogen	11 ± 1.1	9 ± 1.2	17 ± 1.6	19 ± 2.9	17 ± 3.4	11 ± 2.1	9 ± 2.3
Sphingomyelin	276 ±18.4	327 ±20.2	306 ±24.8	236 ± 25.1	*446 ± 26.2	*451 ±27.0	350 ± 23.7
Alkyl Ethers	91 ±10.5	72 ± 5.5	75 ± 9.1	89 ± 14.6	63 ± 12.8	79 ± 7.9	61 ± 8.3

Data represent the mean ± S.E. for each phospholipid.
Box values are significant at the .1% and * values at the 1% level of confidence by Student's t test.

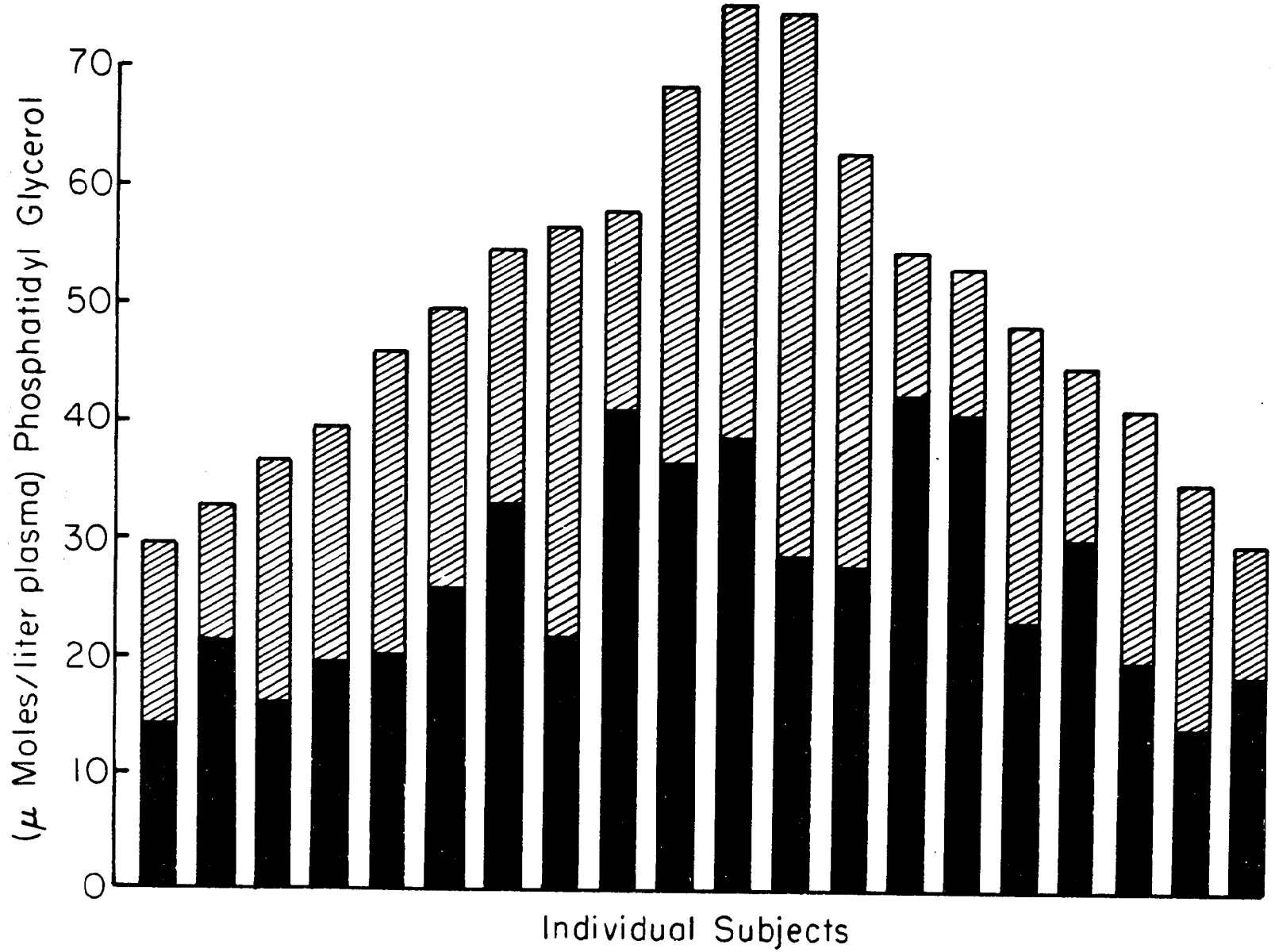

Figure 1. Plasma phosphatidyl glycerol levels in human subjects before and after acceleration to grayout. Solid bars are the individual control levels. Cross hatched bars are the increases after acceleration.

percentage composition of the individual phospholipids obviously implies a different mechanism from that operative when the percentage of the individual phospholipids vary in the face of constant or changing total phospholipid levels. In the stresses examined, the latter condition usually prevailed.

The acceleration stress lasted in the order of seconds and a maximum lapse of 10 to 15 minutes took place between the beginning of the acceleration and the final sampling time. The possibility that the changes observed were related to nutritional factors, obviously, is remote for this particular stress. However, any consideration of the time required for the reversion of the stressed levels of plasma phospholipids to normal might be complicated by phospholipid changes after eating. This was investigated over a 24-hour period in a smaller group of six subjects. The findings are summarized in Figure 2 which portrays the variations in the phospholipids that showed the greatest changes throughout the day. Thus, it is apparent that the stress-induced increments of phosphatidyl glycerol and phosphatidic acid and the decrement in cardiolipin are maintained nearly 3 hours after acceleration, but return to normal levels within 24 hours. This prolonged maintenance of elevated phospholipid levels in the face of the abrupt increase immediately after acceleration suggested the possibility that other factors superimposed their effects onto the effects of acceleration stress. The validity of this postulate became manifest from analyses of the diurnal variations of the plasma phospholipids observed in a volunteer group of 17 subjects on one work day, followed by an acceleration run on the next day. While there is little significant variation in the total phospholipid, (Table III), there is a cyclical variation in other phospholipids throughout the day which show a trough about 9:30 a.m. and a peak in the mid-afternoon. This is illustrated by the bar graphs shown in Figure 3 which plots the concentration of cardiolipin, phosphatidic acid, and phosphatidyl glycerol as the per cent of total phospholipid in the plasma samples taken at 8:00 a.m. (fasting), 9:30 a.m. (after breakfast), 1:00 p.m. (after lunch) and at 3:30 p.m. (at the end of the work day). The following morning a control plasma sample, drawn after breakfast, confirmed the results of the previous day. When acceleration stress was introduced, it superimposed its effect on the normal cycle so that the changes characteristic for the stress became evident. Thus, the increase found with phosphatidyl glycerol exceeded by far the variability during the day. It would appear, then, that the high level of phosphatidyl glycerol obtained immediately after acceleration is a function of the stress per se. The gradual increase in phosphatidyl glycerol between 9 a.m. and 3 p.m. offers a plausible explanation for the sustained high levels for periods of three hours after the initial stress.

The control levels of phosphatidyl glycerol were considerably elevated in the population described in Figure 3 compared to that of the population in Figure 2. Apart from possible unrecognized factors, the major difference between these two groups was that the first group was trained and experienced in the use of the dynamic flight simulator and was accustomed to blood sampling, while the latter group were volunteers who were completely inexperienced and were irritated by the repeated blood sampling.

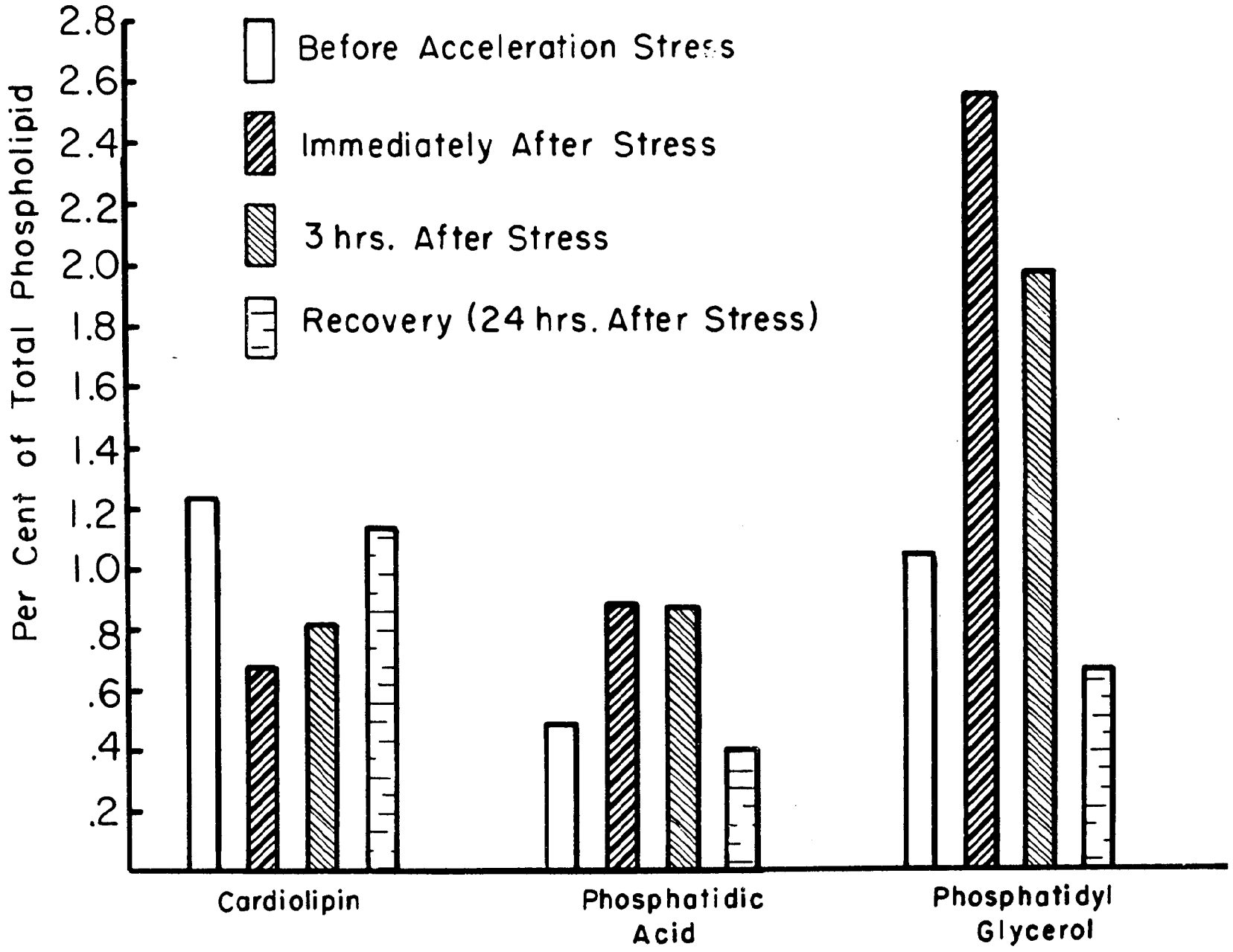

Figure 2. Duration of changes in plasma phospholipids after acceleration to grayout. Concentration changes immediately after and three hours after stress for the three phospholipids are significant at the 5 per cent level of confidence or better. Differences between control and recovery levels are not significant.

TABLE III

DIURNAL VARIATIONS OF PLASMA PHOSPHOLIPIDS
IN NORMAL SUBJECTS

Number of Subjects	17	17	16	16
	Fasting	After Breakfast	After Lunch	End of Work Day
Time	8:00	9:30	13:00	15:30
Phospholipid	μM P/liter plasma			
Total Lipid Phosphorus	2135 ±80.0	2258 ±87.5	2169 ±79.0	2424±103.0
Phosphatidyl Choline	1402 ±64.0	1532 ±69.0	1390 ±57.0	1540 ±74.0
Phosphatidyl Ethanolamine	44 ± 3.4	56 ± 3.5	51 ± 3.4	58 ± 2.3
Phosphatidyl Serine	22 ± 2.0	19 ± 1.4	24 ± 1.9	30 ± 2.2
Cardiolipin	*34 ± 1.6	27 ± 1.9	*36 ± 2.6	38 ± 2.2
Phosphatidic Acid	13 ± 0.8	13 ± 1.3	18 ± 1.8	19 ± 1.8
Phosphatidyl Glycerol	29 ± 1.9	26 ± 2.1	32 ± 1.8	37 ± 2.2
Phosphatidyl Inositide	95 ± 5.6	90 ± 7.0	113 ± 9.1	*118 ± 6.9
Cyclic Glycerophosphoric Acid	*107 ± 5.8	85 ± 4.9	104 ± 6.4	109 ± 7.0
Inorganic Phosphate	15 ± 1.7	15 ± 1.3	17 ± 1.3	20 ± 1.8
Choline Plasmalogen	36 ± 1.7	32 ± 2.3	40 ± 2.8	42 ± 2.2
Ethanolamine Plasmalogen	17 ± 0.9	15 ± 1.4	18 ± 1.2	19 ± 1.3
Serine Plasmalogen	12 ± 0.8	12 ± 1.0	13 ± 0.9	12 ± 0.8
Sphingomyelin	293 ±15.7	321 ±17.9	286 ±14.6	352 ±18.9
Alkyl Ethers	47 ± 2.3	61 ± 6.2	49 ± 4.5	60 ± 5.0

Data represent the mean ± S.E. for each phospholipid.
Indications of statistical significance as in Table II.

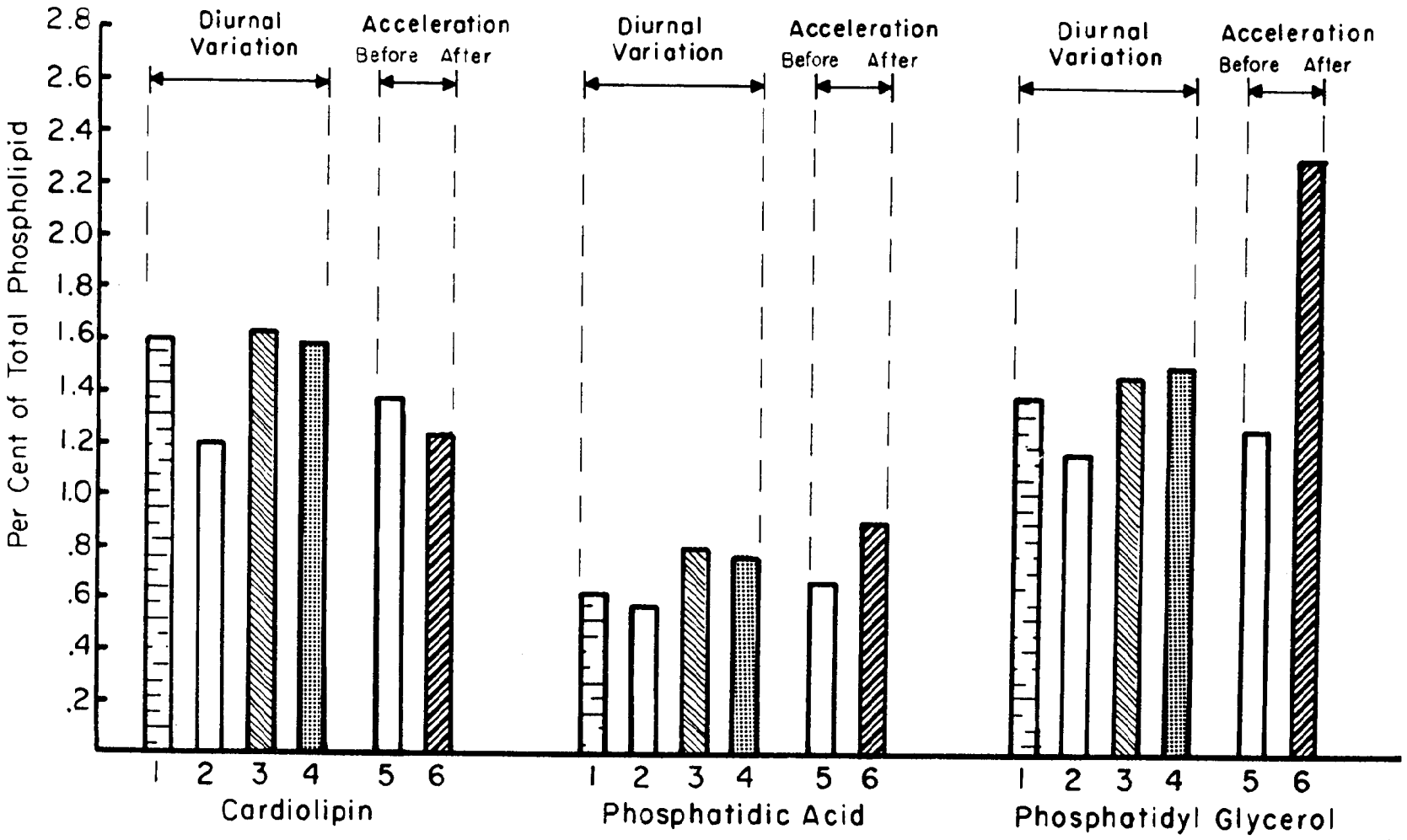

Figure 3. Comparative effects on plasma phospholipids of diurnal variation and acceleration stress on 17 human subjects. 1) fasting 8:00, 2) after breakfast 9:30, 3) after lunch 13:00, 4) end of work day 15:30, 5) after breakfast on the following day, 6) immediately after acceleration on following day. See text for significance.

This inference, that emotional stress may influence the plasma levels of phosphatidyl glycerol, led to the investigation of the phospholipid composition in the plasma of patients with the diagnosis of chronic schizophrenia. The data from twenty patients listed in Table II reveals changes in phosphatidyl glycerol, "phosphatidic acid" and phosphatidyl ethanolamine similar to those obtained after acceleration. The two stressed conditions differed in the quantitative levels of these phospholipids and in the additional elevations in cardiolipin, phosphatidyl serine, and choline plasmalogen found in schizophrenia. In contrast to the results after acceleration, the concentration change of individual phospholipid species in schizophrenia plasma was accompanied by a significant increase in total plasma phospholipid. Comparison of the individual plasma phospholipids in schizophrenia with normal and acceleration plasma phospholipids, after normalizing the data in terms of the per cent of total phospholipid, again revealed significant increments for phosphatidyl glycerol, "phosphatidic acid," phosphatidyl ethanolamine, and phosphatidyl serine, but the changes in cardiolipin and choline plasmalogen became non-significant and obviously were related primarily to the increase in total phospholipid. It appears, then, that in schizophrenia, as in other stressed conditions, the variations of the individual phospholipids reflect the action of regulatory factors which is superimposed on the mechanism controlling the level of the total phospholipid.

For comparison and contrast with the effects of a physical stress like acceleration and a psychic disturbance like schizophrenia, the effect of sleep deprivation, which involves gradual physical and psychic fatigue, was studied. In sleep deprivation, as in acceleration stress, there are significant increments in phosphatidyl glycerol and "phosphatidic acid" and a decrement in cardiolipin with no change in the total phospholipid. The difference in plasma phospholipids found in sleep deprivation and those found in acceleration or schizophrenia, apart from quantitative differences, emanates from the maintenance of normal levels of phosphatidyl ethanolamine and phosphatidyl serine. The phospholipids reported in sleep deprivation were obtained from plasma samples collected at 3 p.m. and represent variations from control levels observed in the same subject 24 hours before the stress and 24 or 48 hours after the stress. These "before stress"and "after recovery from stress" levels were not included in the table since they did not differ significantly from the normal group.

An unusual example of physical and psychic stress which involves "threats to survival" is obtained in the data from naval aviators flying combat missions in Viet Nam. The information obtained is especially interesting since the stress changes observed occur in a population highly selected and specially trained to function within the framework of the stresses endured. Of the 27 aviators whose plasma was sampled during various phases of their tour of duty, the data on 16 studied during the final days of the line combat operation are presented in Table II. This covers the period when they were subjected to the most severe combat missions, and represents the cumulative effects of repeated exposures to combat stress. The results are striking in that qualitatively they resemble the data obtained with the schizophrenic population.

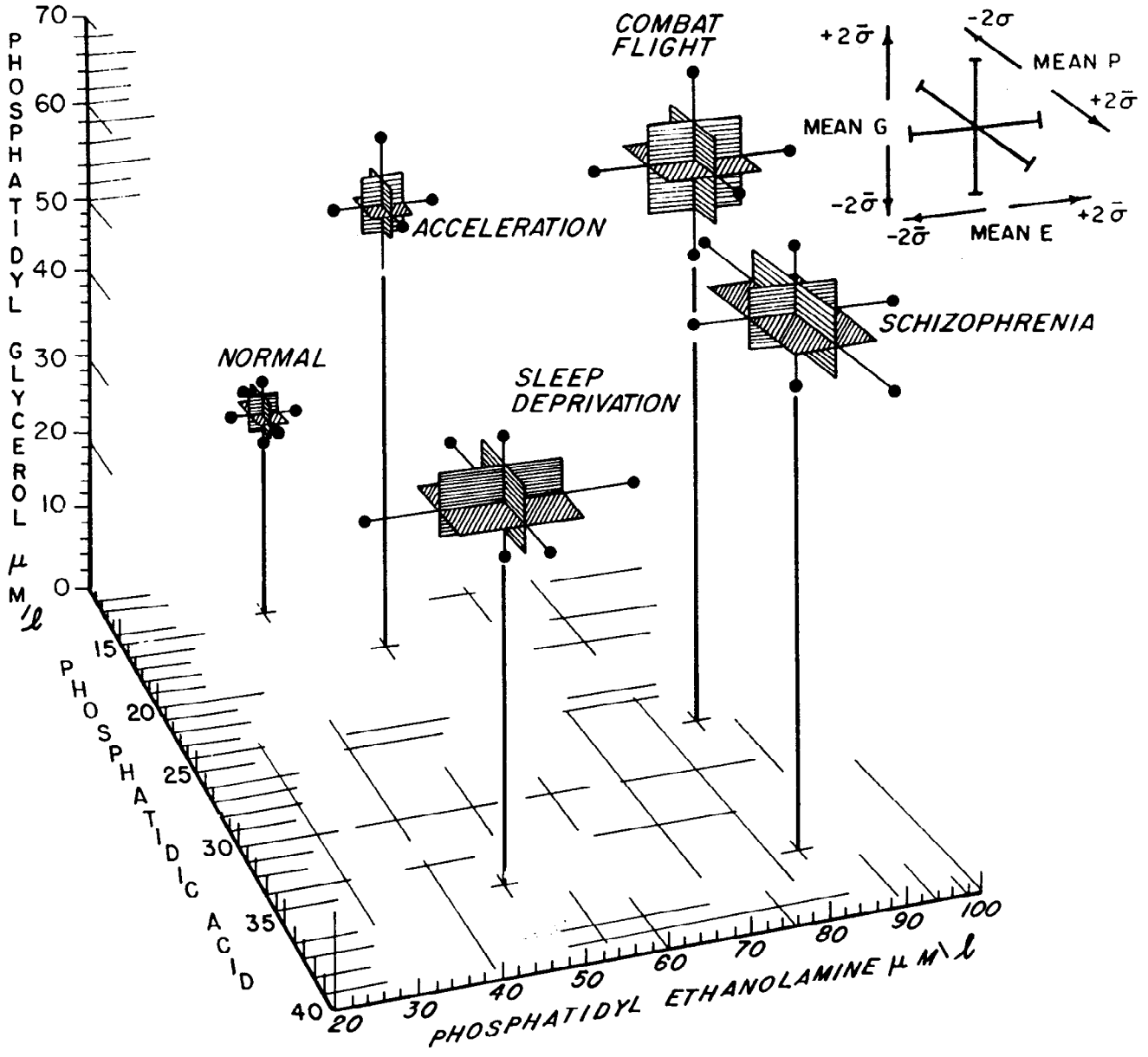

Figure 4. Differentiation of normal and stressed populations by three plasma phospholipids.

Although the similarities of the plasma phospholipid response to stresses with diverse etiologies was by now evident, the data, if it were to be really useful, also should provide some information for possible discrimination of the degree or nature of the stress sustained. In all the physical and psychic stresses, the one common molecular correlate was the marked increase of phosphatidyl glycerol. It was also evident that various other phospholipids become involved in different degrees, depending on the stress. It seemed then that some differential indication of stress etiology might be achieved from the qualitative and quantitative changes in the plasma phospholipids. As a first approach to the characterization of a stress from the biochemical interrelationships of molecular changes, the means plus or minus two standard errors for the three components, phosphatidyl glycerol, "phosphatidic acid," and phosphatidyl ethanolamine, were plotted on a three-dimensional graph (Figure 4). These are represented as intersecting planes which sweep out a volume characteristic of the phospholipid distribution in a given state. In order to portray the statistical nature of these plots and because of the difficulty of constructing normal distribution curves in three dimensions, the first standard error of the mean is represented by the intersecting planes and the second standard error by a single line projection. It is readily seen that the dominating factor in acceleration stress is the great increase in phosphatidyl glycerol. Sleep deprivation is distinguished from acceleration stress by the changes in "phosphatidic acid." Both schizophrenia and combat flight populations are separated from normal, acceleration, or sleep deprivation groups by the significant increments in phosphatidyl ethanolamine. Although there is some overlap, there is also an apparent significant differentiation of the combat group from the schizophrenia group by virtue of quantitative differences in "phosphatidic acid." It is to be emphasized that the separations obtained by this treatment represent a difference in the mean levels of populations rather than one of individual significance.

In the graphical analysis just described, it was possible to attain a measure of discrimination between the normal and stressed populations with three phospholipids. These restrictions were defined by the limitation of graphical visualization to three dimensions. But at least seven of the individual phospholipids analyzed vary significantly with one or another of the stresses considered. If, instead of three reference planes, seven or more dimensions were used, a finer discrimination between the various stresses might be achieved. This multidimensional relationship between the stress etiology and the concentration changes of the plasma phospholipids can be visualized in two dimensions as a spectrum of phospholipid concentration "potentials." These are calculated as the natural log of the ratio of the concentration of each phospholipid to the concentration of the equivalent phospholipid control. The latter concentration (C_{normal}) is obtained from the mean $\log_e$ value of the given phospholipid from a large group of normal subjects. The result is a pattern of values of $\log_e [C_{stress}/C_{normal}]$, at least roughly related to a difference in phospholipid chemical potentials between normal and stress states that "fingerprints" the individual reaction to a stress. In Figure 5 the height of each vertical line represents the

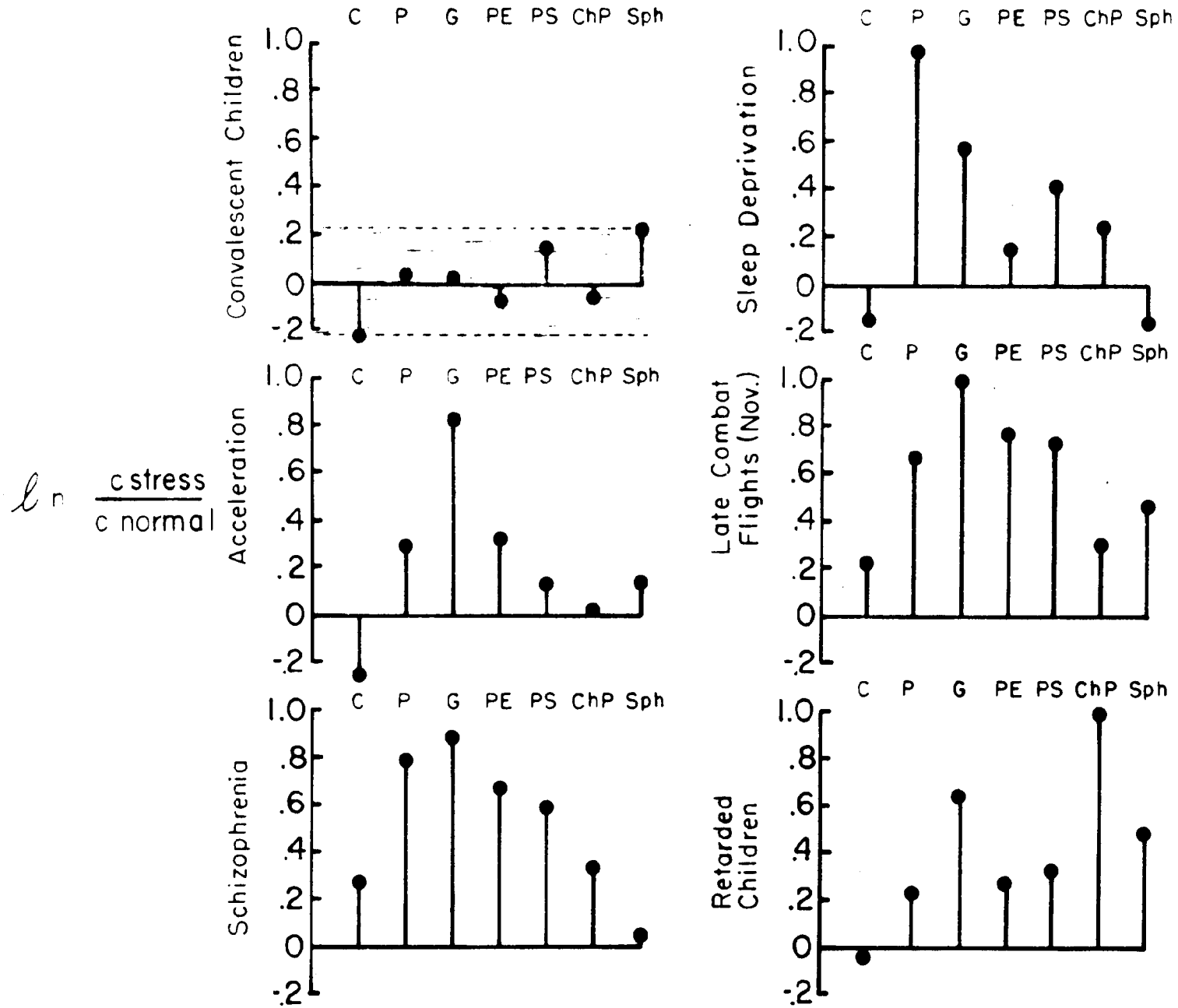

Figure 5. Differentiations of stress in humans from patterns of seven normalized significant plasma phospholipids. C: cardiolipin , P: phosphatidic acid, G: phosphatidyl glycerol, P.E.: phosphatidyl ethanolamine, P.S.: phosphatidyl serine, Ch.P.: choline plasmalogen, Sph: sphingomyelin.

mean $\log_e [C_{stress}/C_{normal}]$ for each of seven phospholipids in the stressed population. The dotted horizontal lines define the range for plus or minus two standard errors of the mean in normal subjects. In the first diagram (a) where a group of convalescent children is compared against young normal adults, the concentration changes are within the error of measurements and no significant difference is manifest. It is seen then that the spectrum of phospholipid "potentials" for a normal individual should approximate a straight line with a height = 0. In sequential order there are listed then patterns for populations subjected to acceleration stress (b), or suffering from schizophrenia (c), exposed to sleep deprivation (d), after combat stress (e) and mentally retarded children (f). Various physical or psychic stresses then are readily associated with a characteristic pattern of changes in phospholipid concentrations.

The two preceding graphical methods of classifying stress from biochemical correlates have an empirical simplicity that show the feasibility of associating physical and psychic stress in the human with molecular changes in the plasma. Substantiation of the findings, however, requires a test of statistical significance. Because several measurements were made on each subject, Student's t test could not be used to test the significance of the differences between means in the various pairs of groups which were compared. The appropriate test must consider all means simultaneously, as well as the intercorrelations among the measurements. Such a test can be based on Hotelling's T^2 statistic which is the multivariate counterpart of Student's t statistic (see, for example, Anderson (12) or Wilks (13)). Hotelling's T^2 can be transformed to an ordinary F ratio so that the test can be performed by reference to standard statistical tables. Just as the t test requires the assumption that the populations being compared have a common variance, the test based on the T^2 statistic requires the assumption that the populations being compared have a common covariance matrix. In the present study, transforming the data to logarithms (base 10) produced covariance matrices which did not differ significantly between pairs of groups, and the statistical analysis has been performed on the logarithms of the original data.

The results of several multivariate tests of significance are given in Table IV. In every case, there are significant differences between stressed and unstressed subjects, and, where comparisons are possible, between subjects under different kinds of stress. Three of the comparisons in Table IV do not show significant differences, but these are comparisons between groups of relatively unstressed subjects which would not be expected to differ significantly. The test on a random split of thirty normal subjects into two groups of fifteen subjects each, was run as a check on the validity of the procedure. Differences among individuals under three different stress conditions are illustrated in Figures 6 and 7. In these figures, the original eight-dimensional space of the measurements has been projected onto a plane in such a way that the "distances" among the centroids of the clusters are maximized. The dividing lines in the figures are the perpendicular bisectors of the sides of the triangle formed by the centroids. By a well known theorem of plane geometry, these three lines meet in a point, and the lines

TABLE IV

F RATIOS FOR SELECTED TWO STRESS COMPARISONS

Stresses Compared	F Ratio	Significance Level
Acceleration: Before-Immediately After	10.31	$P < .01$
Sleep Deprivation: Before-Immediately After	20.53	$P < .05$
Sleep Deprivation: Before-After Recovery	1.21	$P > .05$
Normal-Schizophrenic	10.79	$P < .01$
Normal-Combat Stressed	26.74	$P < .01$
Schizophrenic-Combat Stressed	5.10	$P < .01$
Sleep Deprived-Schizophrenic	8.72	$P < .01$
Sleep Deprived-Combat Stressed	10.27	$P < .01$
Convalescent Children-Mentally Retarded Children	13.22	$P < .01$
Normal-Mentally Retarded Children	15.78	$P < .01$
Normal-Convalescent Children	2.22	$P > .05$
Random Split of Normals	0.56	$P > .05$

Figures 6 and 7. Plots of the adjusted Z indices for individuals under normal and stressed states. In Figure 6, Z_1 separates normals from schizophrenic subjects and pilots in combat; and Z_2 separates the pilots in combat from schizophrenic subjects. The individual points which are misclassified are encircled. In Figure 7, Z_1 separates normal and convalescent children from mentally retarded children; Z_2 shows no significant differentiation between the normal subjects and the convalescent children by discriminant function analysis of the phospholipid data. Essentially the diagrams represent a two-dimensional projection of eight reference planes of phospholipid and cortisol concentrations which characterize the individuals as well as the populations in terms of their reactions to stress.

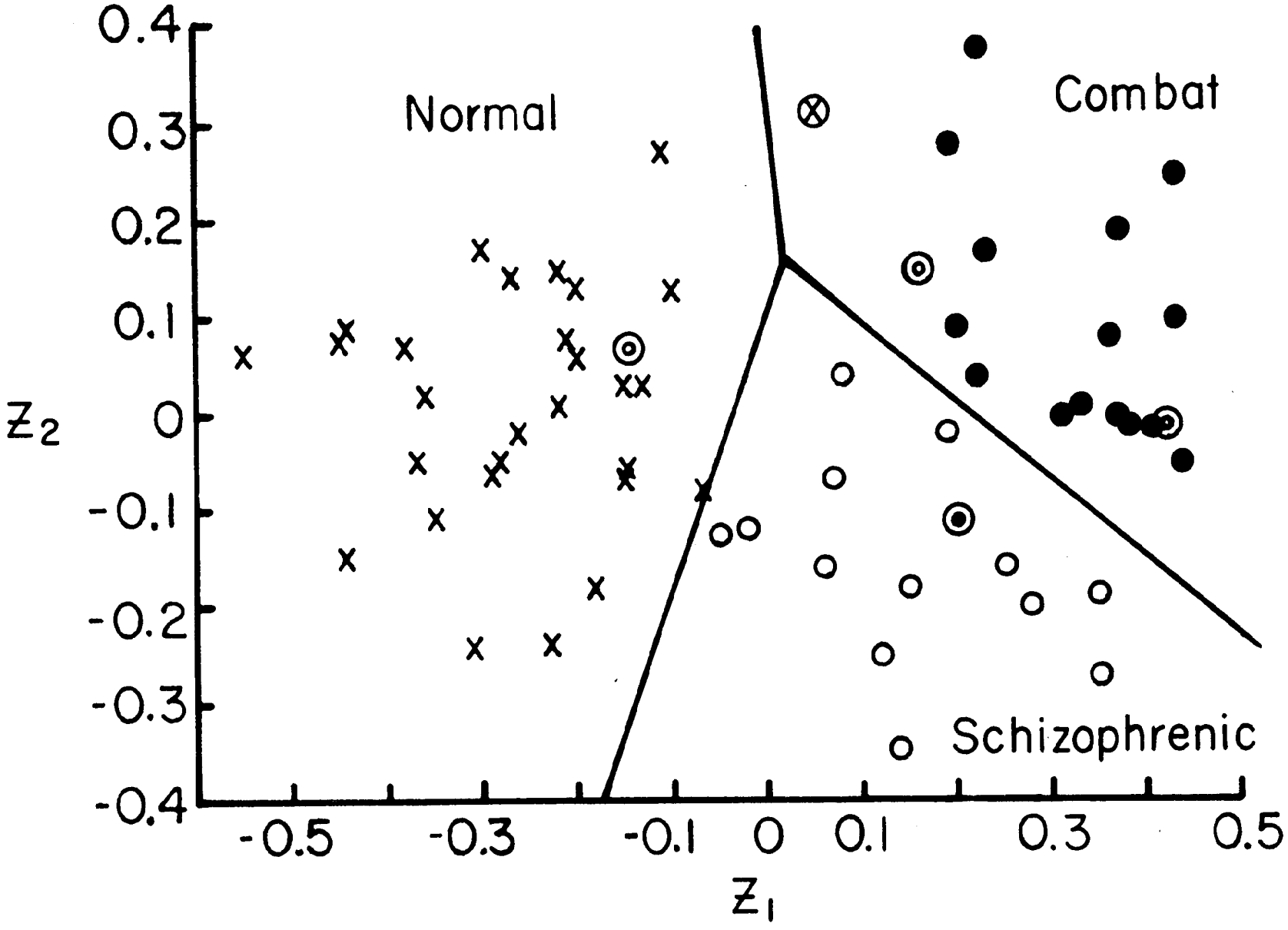
Normal
Combat
Schizophrenic
Z_2
Z_1
0.4
0.3
0.2
0.1
0
-0.1
-0.2
-0.3
-0.4
-0.5
-0.3
-0.1
0
0.1
0.3
0.5

Figure 6

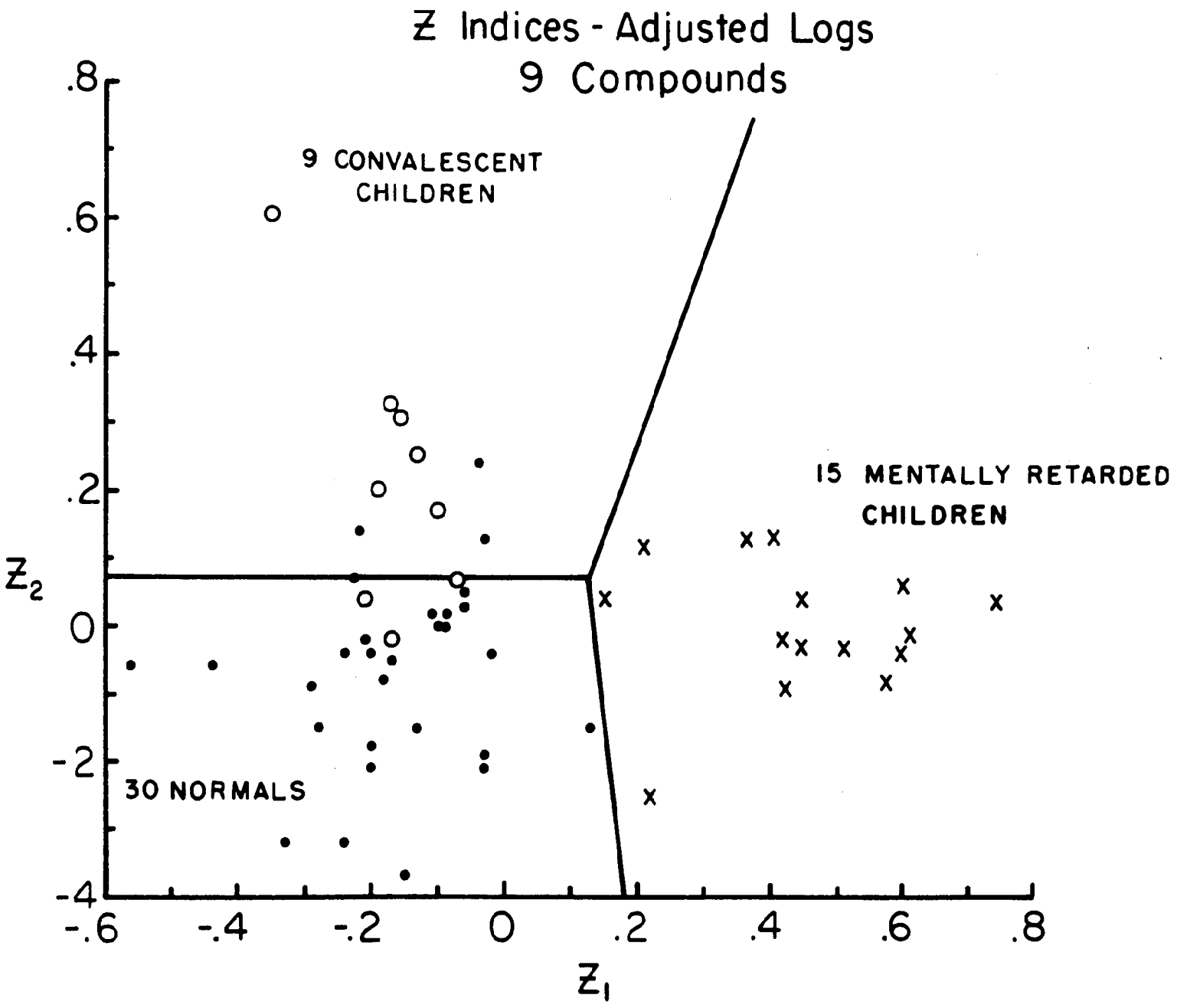
Z Indices - Adjusted Logs
9 Compounds
9 CONVALESCENT CHILDREN
15 MENTALLY RETARDED CHILDREN
30 NORMALS
Z2
Z1
.8
.6
.4
.2
0
-2
-4
-.6
-.4
-.2
0
.2
.4
.6
.8

Figure 7

radiating from the point divide the plane of the projection into three separate regions. If the groups differ, each cluster of points should be concentrated in one of the three regions. The purpose of the projection is to make it possible to "see" differences among clusters of points in a space of more than two dimensions.

The Z indices, Z_1 and Z_2, are computed for each subject by taking two weighted sums of the logarithms of the individual compounds. These sums can be expressed in the form

$$Z_1 = C_{1,1} \log X_1 + C_{1,2} \log X_2 + \ldots + C_{1,8} \log X_8$$

$$Z_2 = C_{2,1} \log X_1 + C_{2,2} \log X_2 + \ldots + C_{2,8} \log X_8.$$

The weights C_{1i} and C_{2i} are determined in such a way that the "distance" among clusters of points in the Z_1, Z_2 plane is maximized while keeping the distance within clusters fixed. When this maximal separation has been achieved, the weights are standardized to the same scale so that the importance of each compound in separating the three groups can be judged. The entire analysis, including the calculation of the F ratios, has been computerized.

DISCUSSION

In the physical and psychic stress reactions cited, phosphatidyl glycerol is unique in the consistent increases of the plasma levels which are common to all the stresses. In contrast, other phospholipids show variable significant changes which permit a correlation with and molecular characterization of the stress. These concentration shifts in individual phospholipids are not a direct consequence of variations in the total phospholipid content, for both increments and decrements of the individual phospholipids can take place with no change or even opposing changes in the level of the total phospholipid content. Whether this represents a concentration effect in the output of a major regulatory factor or whether each phospholipid is uniquely controlled, the results imply the action of some brain center which can interpret certain sensory inputs as "threats to survival" and reacts by mobilizing chemical factors which serve bioenergetically to enhance survival.

Emphasis is placed on acceleration stress because it is a highly reproducible, physically defined entity, whose physiological aspects, such as cardiovascular dynamics and central regulatory factors, are more or less understood. An equivalent state of illusory enlightenment for the biochemical events in acceleration cannot be claimed. Yet, without depreciating the importance of the physiological aberrations of cardiovascular mechanics

which lead to inadequate oxygenation of cellular receptive sites, it is obvious that the final criterion for survival must involve biochemical energetics. Concentration changes in a single metabolite or even a whole class of metabolites do not encompass the enigmas of biological energy. However, by tradition, variations in metabolite concentration have been used as the amplified signal of the action of regulatory or hormonal factors whose identification and quantification may be nearly impossible within the limitations of current theory and technology.

Some indications of possible mechanisms involved in the changes of phosphatidyl glycerol levels in plasma and brain can be inferred from the disappearance of the molecular shifts in the plasma and the enhanced tolerance to acceleration that hypophysectomy causes in the rat. It is apparent that the increase in plasma phosphatidyl glycerol with stress is related to a pituitary function. Whether this represents a hormonally stimulated release of the phospholipid from tissue stores or a direct secretion of the phospholipid is still unknown. MacFarlane and others have given convincing evidence that a number of bacteria produce ortho amino acid esters of phosphatidyl glycerol (14, 15). Phosphatido-peptides have been reported isolated from liver and other tissue (16). It may then not be too remote to postulate a possible release, from the pituitary, of polypeptide esters of phosphatidyl glycerol with hormonal activity. Our finding of higher concentrations of phosphatidyl glycerol in the pituitary than in whole brain, with about twice as high a content in the anterior pituitary as in the posterior pituitary,[1] lends some support to the hypothesis that phosphatidyl glycerol may be involved in the synthesis and transport of pituitary hormones.

Although of hypothetical interest for the plasma changes found in stress, the preceeding speculative explanation does not account for the tissue changes in phosphatidyl glycerol. Preliminary in-vitro experiments indicate that phosphatidyl glycerol accelerates the incorporation of inorganic phosphate into adenosine triphosphate in mitochondria reacting under conditions which support oxidative phosphorylation.[1] Although the mechanism by which this is accomplished has not been clarified, the formation of an energized intermediate from phosphatidyl glycerol, important to the cellular economy, might be assumed. Phosphatidyl glycerol, as analyzed in plasma and tissue, then would represent the degradation product of the energized intermediate. In the normal animal a stress-increased turnover in the utilization of the postulated intermediate could be reflected in elevated levels of the de-energized product. In the hypophysectomized animal some of the metabolic pathways utilizing phosphatidyl glycerol could be inhibited, and the cellular energized intermediate of phosphatidyl glycerol would be available for the primary energy requirements of survival. This rationalization offers at least a speculative explanation for the tissue and plasma changes observed in phosphatidyl glycerol that is consistent with the dramatic increase in tolerance to acceleration found in the hypophysectomized rat.

[1]Polis, B.D., H. Shmukler, H. Schwarz, to be published.

Phosphatidyl glycerol, although present in small quantities in mammalian systems, exists in large concentrations in plant membranes and constitutes the major lipid of many bacteria (17). Significant quantities of phosphatidyl glycerol are found in the chloroplast membranes of E. gracilis grown in the light but are virtually absent from etiolated cells grown in the dark (18). Other related phospholipids have been implicated in both energy transducing reactions and active transport across membranes (19). Also phospholipids have been reported as essential components of both excitatory and receptor systems (20, 21). It would appear then, that even in the face of many gaps in definitive enzymatic pathways, all the available evidence suggest an important role for phosphatidyl glycerol in the energy transducing and synthesis mechanisma of living systems.

One intriguing aspect of the stress studies is the similarity of the phospholipid pattern in chronic schizophrenia and in severe combat stress. Combat stressed subjects and schizophrenics do not differ significantly in the concentrations of the six phospholipids which distinguish schizophrenic subjects from normal. Statistical differentiation between schizophrenia and combat stress is achieved primarily on the differences in sphingomyelin (Table II) and cortisol.[2] Also, schizophrenics *do not* differ from normal subjects and combat stressed subjects *do* differ from normal in the concentrations of these two compounds. A comparison of the plasma phospholipid composition in schizophrenia and in combat fatigue is more marked by similarities than by differences. It is intuitively sensed, and experience confirms, that long exposure to combat can cause extreme psychological disturbance. Yet, from a more objective plane, it would be premature to ascribe the plasma phospholipid variations in a selected, hospitalized, small group of patients as characteristic of the complex behavioral aberrations termed schizophrenia. We interpret these findings to mean not that the combat pilot necessarily is becoming schizophrenic, but that the chronic schizophrenic patient is stressed, and that the schizophrenic brain, like the combat pilot's brain, is interpreting its environment as a threat to survival and is reacting biochemically to meet this threat.

It has been shown earlier that the plasma phospholipids revert rapidly to normal levels with rest in all the stresses observed except those of combat and schizophrenia. In combat stress there was a slow remission toward normal after removal from the combat area and return home. In *chronic* schizophrenia the plasma phospholipid pattern tended to remain fixed, as if indicative of a locked stress pattern with the normal regenerative pathways either exhausted or blocked. An experimental attack on this regenerative mechanism in the human presents almost insurmountable difficulties. However, the similarities of the phospholipid variations in the stressed human and the stressed rat, taken in conjunction with the correlations of changes in plasma with comparable changes in the tissue of the stressed rat, offer an experimental approach to cellular control mechanisms that have still resisted any significant resolution.

---

[2]Noval, J. and T. Post, to be published.

REFERENCES

1. Schwarz, H.P., L. Dreisbach, E. Polis, B.D. Polis, E. Soffer. 1965. Effect of whole body X-ray irradiation on phospholipids of rat liver particulate fractions. *Arch. Biochem. Biophys. 111:* 422.

2. Polis, B.D. 1961. Hormonal determinants of mammalian tolerance to acceleration stress. *J. Applied Physiol. 16:* 211.

3. Dawson, R.M.C. 1962. Improvements in the method of determining individual phospholipids in a complex mixture by successive chemical hydrolyses. *Biochem. J. 84:* 497.

4. Wells, M.A., J.C. Dittmer. 1963. The use of sephadex for the removal of nonlipid contaminants from lipid extracts. *Biochemistry 2:* 1259.

5. Dawson, R.M.C. 1960. A hydrolytic procedure for the identification and estimation of individual phospholipids in biological samples. *Biochem. J. 75:* 45-53.

6. Bartlett, G.R. 1959. Phosphorus Assay in Column Chromatography. *J. Biol. Chem. 234:* 466.

7. Sipple, W.C., B.D. Polis. 1961. The electrocardiogram as an indicator of acceleration stress. *IRE Trans. on Bio-Med. Electr. BME-8 (3):* 189.

8. Crosbie, R. 1967. Performance characteristics of AMRD centrifuge for humans. U.S. Naval Air Development Center, Aerospace Medical Research Dept., Johnsville, Warminster, Pa. NADC-MR-6721.

9. York, E., N.J. Colosi, and J.H. Roediger. 1966. Human biochemical parameters of acceleration stress. U.S. Naval Air Development Center, Aerospace Medical Research Dept., Johnsville, Warminster, Pa. NADC-MR-6603.

10. Austin, F.H., T.J. Gallagher, C.A. Brictson, B.D. Polis, D.E. Furry, C.E. Lewis. 1967. Aeromedical monitoring of naval aviators during aircraft carrier combat operations. *Aerospace Med. 38 (6):* 593.

11. Entenman, I.C., R.A. Neve, H. Supplee, and C.A. Olmstedt. 1955. Effects of X-irradiation on lipide metabolism. I. Plasma phospholipide levels in several species. *Arch. Biochem. Biophys. 59:* 97.

12. Anderson, T.W. 1958. Introduction to Multivariate Statistical Analysis. John Wiley and Sons, New York.

13. Wilks, S.S. 1962. Mathematical Statistics. John Wiley and Sons, New York.

14. MacFarlane, M.G. 1964. Phosphatidylglycerols and lipoamino acids. *Adv. Lipid Res. 2:* 91-125.

15. Houtsmuller, U.M.T., L.L.M. van Deenen. 1963. Identification of bacterial phospholipid as an O-ornithine ester of phosphatidyl glycerol. *Biochim. Biophys. Acta, 70:* 211-213.

16. Huggins, C.G., D.V. Cohn. 1959. Studies concerning the composition, distribution, and turnover of phosphorus in a phosphatido-peptide fraction from mammalian tissue. *J. Biol. Chem. 234:* 257.

17. Houtsmuller, U.M.T., L.L.M. van Deenen. 1965. On the amino acid esters of phosphatidyl glycerol from bacteria. *Biochim. Biophys. Acta, 106:* 564-576.

18. Haverkate, F., L.L.M. van Deenen. 1965. Studies of the phospholipids of photosynthetic microorganisms. *I. Koninkl. Ned. Akad, Wetenschap, Proc. Ser. B. 68:* 141-153.

19. Van Deenen, L.L.M. 1965. Phospholipids and biomembranes. *In* Progress in Chemistry of Fats and Other Lipids. R.T. Holman, Ed. Pergamon Press, Oxford. 8 (1): 88.

20. Dickstein, S., F.G. Sulman. 1965. Function of phospholipids in receptors. *Biochem. Pharmacol. 14:* 739.

21. Tanaka, R., K.P. Strickland. 1965. Role of phospholipid in the activation of Na^+, Ka^+-activated adenosine triphosphatase of beef brain. *Arch. Biochem. Biophys. 111:* 583.

APPENDIX A

EFFECT OF ACCELERATION ON PLASMA PHOSPHOLIPIDS OF NORMAL AND HYPOPHYSECTOMIZED RATS

	Total Lipid Phosphorus µM P/liter	Lecithin	Phosphatidyl Ethanolamine	Phosphatidyl Serine	Cardiolipin	Phosphatidic Acid	Phosphatidyl Glycerol	Phosphatidyl Inositide	Cyclic Glycero-phosphoric Acid	Inorganic Phosphorus	X_2	Choline Plasmalogen	Ethanolamine Plasmalogen	Serine Plasmalogen	Unknown Plasmalogen	Sphingomyelin	Acid Labile Unknowns	Alkyl Ethers	Number of Experiments	Number of Rats
		Per Cent of Total Phospholipid																		
Normal Control																				
Mean	2291	76.98	1.85	.91	1.16	.60	.94	4.48	3.60	.52	.62	1.04	.64	.43	.34	7.87	.67	1.41	11	27
S.D. ±	776	2.36	.22	.26	.24	.20	.30	.85	.95	.26	.16	.22	.24	.24	.34	1.40	.36	.92		
S.E. ±	234	.71	.07	.08	.07	.06	.09	.25	.29	.08	.05	.07	.07	.07	.10	.42	.11	.28		
Normal Accelerated																				
Mean	1633	71.98	1.62	.85	.95	1.35	2.35	5.64	5.90	.58	.68	1.37	.67	.71	.66	6.72	.69	2.99	4	20
S.D. ±	393	8.19	.68	.47	.64	.97	.58	.79	2.45	.35	.36	.85	.51	.47	.36	1.43	.21	1.64		
S.E. ±	197	4.09	.34	.24	.32	.49	.29	.39	1.23	.17	.18	.42	.25	.24	.18	.71	.10	.82		
Hypophysectomized Control																				
Mean	1597	72.24	1.78	.48	1.34	.76	.98	6.94	4.04	.62	1.19	.83	.35	.42	.32	9.90	.53	3.34	3	32
S.D. ±	599	5.59	.62	.15	.21	.45	.59	1.01	3.18	.23	.26	.18	.16	.15	.05	2.39	.09	2.30		
S.E. ±	346	3.22	.36	.09	.12	.26	.34	.58	1.84	.13	.15	.10	.09	.08	.03	1.38	.05	1.33		
Hypophysectomized Acceleration																				
Mean	1465	71.57	1.88	.55	1.27	.71	1.04	6.58	5.69	.51	.57	1.24	.64	.43	.47	10.27	1.08	1.45	3	45
S.D. ±	509	6.32	.33	.28	1.29	.31	.23	.72	1.16	.52	.08	.73	.26	.26	.13	1.32	.38	.81		
S.E. ±	294	3.65	.19	.16	.75	.18	.13	.42	.67	.30	.04	.42	.15	.15	.07	.76	.22	.47		
□	P(t)	<.001																		
—	P(t)	<.05																		

THE EFFECT OF ACCELERATION ON BRAIN PHOSPHOLIPIDS FROM NORMAL AND HYPOPHYSECTOMIZED RATS

	Total Lipid Phosphorus µM P/g dry residue	Lecithin	Phosphatidyl Ethanolamine	Phosphatidyl Serine	Cardiolipin	Phosphatidic Acid	Phosphatidyl Glycerol	Phosphatidyl Inositide	Cyclic Glycero-phosphoric Acid	Inorganic Phosphorus	X_2	Choline Plasmalogen	Ethanolamine Plasmalogen	Serine Plasmalogen	Sphingomyelin	Alkyl Ethers	Number of Experiments	Number of Brains	
								Per Cent of Total Phospholipid											
Normal Control																			
Mean	491.4	33.1	18.3	11.5	2.2	3.1	1.5	5.2	3.8	.7	1.0	2.9	6.7	1.1	8.2	2.0	8	10	
S.D. ±	35.6	.80	.68	.72	.19	.33	.18	.47	.73	.14	.39	.37	1.82	.42	1.68	.41			
S.E. ±	12.6	.28	.24	.26	.07	.12	.06	.17	.26	.05	.14	.13	.64	.15	.59	.14			
Normal Accelerated																			
Mean	520.5	34.4	14.2	10.7	2.5	4.1	2.5	7.4	6.3	1.0	1.5	1.9	5.5	.5	6.8	2.4	3	11	
S.D. ±	44.4	8.05	4.72	1.70	.20	1.26	2.11	3.13	3.09	.47	1.06	.21	7.45	.24	1.41	.52			
S.E. ±	25.6	4.65	2.72	.98	.11	.73	1.22	1.81	1.78	.27	.61	.12	4.30	.14	.82	.30			
Hypophysectomized Control																			
Mean	562.3	35.4	14.4	11.0	2.4	3.0	1.9	6.0	5.4	1.1	1.5	2.0	8.0	.4	8.3	1.8	3	15	
S.D. ±	28.5	7.39	4.27	1.50	.26	.10	.49	1.26	1.75	.49	.95	.53	4.65	.12	1.70	.79			
S.E. ±	16.4	4.27	2.47	.87	.15	.06	.28	.73	1.01	.28	.55	.31	2.69	.07	.98	.46			
Hypophysectomized Accelerated																			
Mean	601.8	34.2	12.2	8.9	2.7	3.7	3.4	6.0	6.3	1.2	1.6	1.7	10.1	.6	7.5	1.9	4	22	
S.D. ±	69.4	4.91	3.06	1.48	.41	.81	.79	1.48	1.10	3.87	.37	.13	5.23	.29	1.37	.60			
S.E. ±	34.7	2.45	1.53	.74	.21	.41	.39	.74	.55	.19	.19	.06	2.62	.14	.69	.30			
	P(t)	<.001																	
	P(t)	<.05																	

THE EFFECT OF ACCELERATION ON LIVER PHOSPHOLIPIDS FROM NORMAL RATS

	Total Lipid Phosphorus μM P/g dry residue	Lecithin	Phosphatidyl Ethanolamine	Phosphatidyl Serine	Cardiolipin	Phosphatidic Acid	Phosphatidyl Glycerol	Phosphatidyl Inositide	Cyclic Glycero-phosphoric Acid	X_2	Inorganic Phosphorus	Choline Plasmalogen	Ethanolamine Plasmalogen	Serine Plasmalogen	Sphingomyelin	Alkyl Ethers	Number of Livers
						Per Cent of Total Phospholipid											
Control																	
#1	116.1	54.2	18.1	4.0	5.1	1.3	1.2	9.8	7.4	1.3	1.1	.7	.4	0	2.0	1.5	
2	144.5	52.0	16.4	3.7	5.2	1.5	1.1	7.6	10.8	1.5	1.4	.6	.2	0	1.9	1.1	
3	134.0	48.8	17.9	3.5	4.2	.9	1.0	8.3	4.1	1.0	.7	.3	.4	0	2.2	.6	
4	111.5	49.3	24.0	3.3	5.1	.8	.7	8.8	5.7	.8	.7	1.3	.2	0	2.2	.6	
5_1	111.4	47.5	21.3	3.4	4.8	1.0	.7	8.7	5.5	.9	1.0	.2	.6	.4	3.6	1.2	
6_1	144.1	49.7	22.4	2.8	4.0	.8	.6	7.3	4.7	.8	.7	.7	.5	.3	4.2		
5_2-unrestrained	102.4	49.6	22.7	3.5	5.6	1.5	1.2	12.0	6.4	2.1	1.3	.7	.3	0	2.4	1.3	
6_2-unrestrained	100.0	50.0	16.7	4.1	5.4	1.8	1.6	10.2	9.0	2.3	1.4	.6	.2	0	1.7	1.1	
Mean	120.5	50.1	19.9	3.5	4.9	1.2	1.0	9.1	6.7	1.3	1.0	.6	.4	.1	2.5	1.1	8
S.D. ±	17.91	2.07	2.99	.41	.56	.38	.34	1.53	2.26	.59	.31	.33	.15	.17	.89	.34	
S.E. ±	6.33	.73	1.06	.15	.20	.13	.12	.54	.80	.21	.11	.11	.05	.07	.31	.13	
Accelerated																	
1	160.7	51.0	18.1	3.7	4.5	2.5	2.9	8.4	6.3	1.5	1.3	.4	.1	.1	2.0	.5	
2	147.2	50.2	17.1	3.3	4.6	2.0	2.0	9.4	5.8	1.0	.7	.1	.3	.2	2.0	.4	
3	179.8	51.6	20.9	3.0	5.1	2.1	2.3	9.1	6.5	.8	1.0	.8	.4	.1	1.8	.3	
4	120.5	48.0	20.9	3.3	4.2	1.8	2.6	8.7	5.2	.7	.7	.3	.5	.1	1.9	.6	
5	124.4	49.3	20.8	3.3	4.1	1.2	1.6	9.2	5.7	1.0	1.0	.3	.6	.3	2.0	.8	
6	131.2	51.6	16.5	3.6	4.5	1.6	1.8	9.4	4.6	1.3	.8	.8	.1	.2	2.5	.9	
Mean	144.0	50.3	19.1	3.4	4.5	1.9*	2.2	9.0	5.7	1.1	.9	.5	.3	.2	2.0	.6	6
S.D. ±	23.11	1.43	2.05	.25	.35	.45	.49	.40	.70	.30	.23	.29	.21	.08	.24	.23	
S.E. ±	9.44	.58	.84	.10	.14	.18	.20	.16	.29	.12	.09	.12	.08	.03	.10	.09	
□	P(t)	<.001															
*	P(t)	<.01															
—	P(t)	<.05															

THE EFFECT OF ACCELERATION ON LIVER PHOSPHOLIPIDS FROM HYPOPHYSECTOMIZED RATS

	Total Lipid Phosphorus µM P/g dry residue	Lecithin	Phosphatidyl Ethanolamine	Phosphatidyl Serine	Cardiolipin	Phosphatidic Acid	Phosphatidyl Glycerol	Phosphatidyl Inositide	Cyclic Glycero-phosphoric Acid	X_2	Inorganic Phosphorus	Choline Plasmalogen	Ethanolamine Plasmalogen	Serine Plasmalogen	Sphingomyelin	Alkyl Ethers	Number of Livers
	Per Cent of Total Phospholipid																
Control																	
2	136.0	48.9	21.7	2.3	4.9	.9	1.3	10.2	7.7	1.1	.8	.4	.3	.1	.8	.7	
3	117.5	48.1	22.2	3.6	4.6	1.0	1.3	10.1	4.6	2.0	1.2	.6	.2	.2	2.1	1.6	
4	120.1	54.6	15.9	2.1	4.6	1.3	1.4	6.9	6.0	2.1	1.0	1.1	.5	.1	4.8	.5	
8	129.0	52.6	15.0	1.7	4.2	1.9	1.6	7.0	8.8	2.0	1.2	.7	.4	.1	4.9	.7	
9	142.3	50.4	20.8	3.1	4.7	1.5	1.3	9.0	6.2	1.2	.7	.4	.4	.1	3.6	.6	
10	129.2	49.4	21.1	4.1	4.1	1.2	.9	9.2	4.5	.8	.7	.5	.5	.1	4.9	1.6	
Mean	129.0	50.7	19.5	2.8	4.5	1.3	1.3	8.7	6.3	1.5	.9	.6	.4	.1	3.5	1.0	6
S.D. ±	9.35	2.47	3.15	.94	.31	.36	.23	1.40	1.70	.56	.23	.26	.12	.002	1.72	.51	
S.E. ±	3.82	1.01	1.29	.38	.12	.15	.09	.61	.69	.23	.10	.11	.05	.04	.70	.21	
Accelerated																	
2	127.9	48.0	17.7	3.3	5.2	2.2	2.4	9.2	4.9	1.3	1.7	.7	.5	.4	4.5	.8	
3	139.3	49.1	20.9	3.1	5.2	1.6	2.0	8.8	5.9	1.5	.8						
4	154.5	49.7	24.4	3.1	4.8	1.4	1.8	8.4	4.5	.9	.6						
5	140.6	49.5	22.4	3.2	4.2	1.9	1.9	7.4	6.2	.8	1.0	.7	.6	.2	2.7	.6	
7	127.9	47.0	18.7	3.1	3.9	1.7	2.0	9.8	7.5	1.9	.9	1.2	.8	.2	3.5	.4	
8	132.7	46.8	19.2	2.9	4.4	2.1	2.0	8.4	6.9	1.5	.7	.8	.6	.1	5.4	.4	
10	172.0	51.5	21.8	2.6	4.2	1.4	1.6	7.7	5.1	1.1	1.0	.5	.5	.1	3.0	1.3	
Mean	142.1	48.8	20.7	3.0	4.6	1.8	2.0	8.5	5.9	1.3	1.0	.8	.6	.2	3.8	.7	7
S.D. ±	16.07	1.66	2.35	.23	.52	.32	.24	.83	1.10	.38	.36	.26	.12	.12	1.12	.37	
S.E. ±	6.07	.63	.89	.09	.20	.12	.09	.31	.42	.15	.14	.12	.05	.05	.50	.17	
□	P(t)	<.001															
*	P(t)	<.01															
—	P(t)	<.05															

THE EFFECT OF WHOLE BODY IRRADIATION ON THE PHOSPHOLIPID COMPOSITION OF RAT PLASMA

	Total Lipid Phosphorus µM P/liter	Lecithin	Phosphatidyl Ethanolamine	Phosphatidyl Serine	Cardiolipin	Phosphatidic Acid	Phosphatidyl Glycerol	Phosphatidyl Inositide	Cyclic Glycero-phosphoric Acid	Alkali Labile Unknowns	Choline Plasmalogen	Ethanolamine Plasmalogen	Serine Plasmalogen	Unknown Plasmalogen	Sphingomyelin	Acid Labile Unknowns	Alkyl Ethers
							Per Cent of Total Phospholipid										
Control																	
Experiment Number*																	
1	1433	65.7	1.4	1.0	1.1	1.1	1.1	5.4	7.9	1.0	1.4	.6	.6	0	11.7	0	
2	1110	65.6	1.1	.6	1.1	1.1	1.0	4.0	5.4	7.3	1.6	0	.3	.7	10.4	0	
3	2130	68.3	2.4	1.6	1.4	1.1	1.2	3.8	10.1	3.1					6.4		
4	1750	67.0	2.3	2.2	1.4	1.0	.8	6.3	3.8	1.1	1.5	.6	.1	.2	7.0	.7	2.0
5	1107	63.7	2.2	1.2	1.0	1.2	.9	4.9	6.5	1.7	1.2	1.1	.9	.8	9.7	.7	2.3
6	1267	67.1	.6	.6	.7	.9	1.1	3.3	4.7	5.9	.8	.3	.3	.3	7.9	.4	5.1
7	1 5	74.9	1.7	.4	.5	.8	1.0	3.5	4.1	1.2	.8	.4	.3	.6	6.8	.3	2.7
8	1580	57.9	3.5	2.5	2.0	1.6	1.1	6.4	7.7	3.3	.7	.4	.2	.1	6.5	.9	5.5
Mean	1517	66.4	1.9	1.3	1.1	1.1	1.0	4.7	6.3	3.1	1.6	.4	.3	.3	8.3	.4	2.2
S.D. ±	358	4.79	.90	.78	.53	.24	.13	1.18	2.19	2.38	.53	.36	.29	.32	2.03	.24	1.65
S.E. ±	127	1.69	.32	.27	.19	.08	.05	.42	.77	.84	.19	.13	.10	.11	.72	.09	.58
Irradiated																	
Experiment Number*																	
1	1414	60.5	3.4	1.1	1.4	1.8	2.2	3.4	9.3	4.5	1.4	.8	0	.4	9.7		
2	1459	60.3	1.6	1.4	1.2	1.3	1.9	3.5	6.3	6.3	2.6	0	.5	.8	12.8		
3	2474	71.0	2.3	.7	.9	1.0	1.5	3.4	5.6	4.4					8.9		
4	1963	72.6	1.6	.7	1.0	1.2	1.1	4.5	5.4	1.9	.8	.7	.1	.2	5.2	.6	1.9
5	1983	70.2	2.2	.8	1.2	1.6	1.9	4.6	4.5	1.5	1.0	.7	.4	.2	7.0	.3	2.0
6	1605	70.3	.7	.3	1.7	1.4	2.1	4.4	5.0	1.3	.8	.5	.3	.1	6.3	1.4	3.3
7	2404	75.4	3.2	.4	.5	1.7	2.0	3.1	.8	.8	.7	.2	.2	.2	8.6	.2	1.9
8	2486	68.7	1.9	1.3	1.1	1.8	1.9	3.7	3.1	2.3	1.2	.8	.4	.9	8.6	.9	1.5
Mean	1986	68.6	2.1	.8	1.1	1.5	1.8	3.8	5.0	2.9	1.0	.5	.2	.3	8.4	.4	1.9
S.D. ±	434	5.45	.88	.40	.36	.30	.36	.59	2.46	1.95	.58	.35	.19	.31	2.33	.49	.85
S.E. ±	153	1.93	.31	.14	.13	.11	.13	.21	.87	.69	.21	.12	.07	.11	.82	.17	.30
□	P(t)	<.001															
—	P(t)	<.02															
*	Each experiment represents a pool of plasma from three rats.																

THE EFFECT OF WHOLE BODY IRRADIATION ON THE PHOSPHOLIPID COMPOSITION OF RAT LIVERS

	Total Lipid Phosphorus µM P/liter	Lecithin	Phosphatidyl Ethanolamine	Phosphatidyl Serine	Cardiolipin	Phosphatidic Acid	Phosphatidyl Glycerol	Phosphatidyl Inositide	Cyclic Glycero-phosphoric Acid	Alkali Labile Unknowns	Choline Plasmalogen	Ethanolamine Plasmalogen	Serine Plasmalogen	Unknown Plasmalogen	Sphingomyelin	Acid Labile Unknowns	Alkyl Ethers	
		Per Cent of Total Phospholipid																
Control Rat																		
1	4361	53.6	20.1	3.6	5.0	2.7	1.7	8.4	8.0	1.6	.2	.3	.03	.2	2.5	.3	1.4	
2	4704	51.1	21.0	3.6	5.4	1.5	2.3	5.7	5.8	2.1	.9	1.0	.4	.2	2.0	.2	.7	
3	4413	50.6	18.5	4.0	4.6	1.3	.8	6.2	8.9	1.0	.3	.8	.05	.05	3.1	.4	1.2	
4	2647	49.0	22.5	3.6	5.0	1.7	1.6	5.7	5.6	.7	.3	.7	.4	.2	2.6	.1	.7	
5	3827	48.7	20.4	3.8	4.9	1.2	1.4	5.0	3.8	1.5	.5	.9	.2	.2	2.4	.2	.9	
6	3989	51.9	22.2	4.2	5.3	1.3	1.9	8.4	2.1	2.5	.6	.9	.3	.3	2.6	.4	.7	
7	3597	53.5	22.2	3.2	5.0	2.0	1.5	6.3	5.1	1.2	.3	.4	.2	.4	3.2	.2	.6	
8	4151	51.1	26.8	3.6	5.0	1.6	1.5	6.0	6.1	1.0	.2	.7	.1	.3	3.1	.4	1.0	
9	3990	52.3	25.1	4.4	5.1	1.8	1.7	7.3	7.2	1.0	.5	.8	.2	.1	3.7	.2	.5	
10	4286	50.5	23.8	3.7	3.3	.8	1.7	6.9	5.3	.8	.6	.9	.6	.1	4.3	.2	.5	
11	3597	49.9	23.4	4.2	5.0	1.7	1.7	7.2	6.5	1.5	.4	.8	.2	.04	4.0	.1	.7	
Mean	3957	51.1	22.4	3.8	4.9	1.6	1.6	6.6	5.8	1.4	.4	.7	.2	.2	3.0	.2	.8	
S.D. ±	584	1.62	2.37	.35	.56	.49	.36	1.10	1.88	.56	.21	.2	.17	.11	.72	.11	.29	
S.E. ±	185	.49	.71	.11	.17	.15	.11	.33	.57	.17	.06	.06	.05	.03	.22	.03	.09	

THE EFFECT OF WHOLE BODY IRRADIATION ON THE PHOSPHOLIPID COMPOSITION OF RAT LIVERS

Irradiated Rat No.	Total Lipid Phosphorus µ Moles	Lecithin	Phosphatidyl Ethanolamine	Phosphatidyl Serine	Cardiolipin	Phosphatidic Acid	Phosphatidyl Glycerol	Phosphatidyl Inositide	Cyclic Glycero-phosphoric Acid	Alkali Labile Unknowns	Choline Plasmalogen	Ethanolamine Plasmalogen	Serine Plasmalogen	Unknown Plasmalogen	Sphingomyelin	Acid Labile Unknowns	Alkyl Ethers
		Per Cent of Total Phospholipid															
1	4035	53.4	19.4	3.5	5.2	3.9	2.6	7.4	7.4	2.8	.2	.2	.1	.2	3.3	.1	1.0
2	4396	50.7	18.9	3.4	4.7	1.8	3.1	5.0	6.2	2.2	.3	.8	.5	.2	2.0	.2	1.0
3	4601	54.5	18.6	3.2	4.0	1.6	2.3	4.2	4.4	1.1	.1	.5	.1	.1	2.3	.3	1.2
4	4929	50.6	22.6	3.3	4.0	1.6	.9	6.6	3.7	1.3	.3	.6	.3	.1	3.3	.3	.9
5	3844	54.5	22.9	3.8	4.5	1.7	2.8	7.9	3.6	1.6	.6	.6	.2	.2	2.4	.2	1.1
6	3429	54.9	15.3	3.4	4.3	2.0	1.2	4.7	5.9	1.2	.05	1.1	.4	.04	4.1	.6	1.3
7	4612	54.2	21.7	3.7	4.3	1.6	1.6	6.5	6.2	1.5	.4	.3	.1	1.0	3.1	.1	.8
8	4063	46.1	15.3	3.3	4.0	2.3	1.7	5.3	5.1	.9	.4	.2	.2	.2	3.1	.2	1.3
9	3948	48.9	21.0	3.6	4.8	1.4	1.7	7.6	5.4	2.4	.4	.9	.1	.03	1.5	.3	.4
10	4831	47.8	24.1	4.8	5.0	1.7	2.5	8.4	6.1	2.4	.9	1.0	.2	.4	2.1	.1	1.2
11	4574	40.9	22.4	4.3	4.8	1.5	1.6	8.8	4.4	4.8							
12	4653	55.6	19.8	4.4	4.4	1.6	2.4	8.9	5.4	5.9							
13	4280	48.7	21.5	4.4	4.2	1.7	2.6	10.9	5.4	9.0							
14	4271	50.0	22.2	3.8	4.4	1.6	2.1	8.2	6.6	2.3							
15	3902	47.6	20.0	3.9	4.4	1.8	2.8	8.2	6.0	7.4							
16	4482	46.8	21.1	3.6	4.4	1.4	1.8	7.4	5.0	3.5							
17	3440	50.1	20.6	4.7	4.8	1.2	1.8	6.5	5.4	4.2							
18	4760	53.5	20.7	3.7	4.4	1.8	2.4	9.4	6.3	4.4							
19	4929	48.5	19.6	2.3	4.6	1.8	2.0	8.7	8.0	1.5							
20	3429	52.0	20.1	5.3	4.8	2.0	2.1	8.8	6.2	2.0							
Mean	4270	50.9	20.4	3.8	4.5	1.8	2.1	7.5	5.6	3.1	.3	.6	.2	.2	2.7	.2	1.0
S.D. ±	491	2.98	2.24	.68	.33	.55	.57	1.72	1.11	2.22	.16	.33	.14	.29	.78	.16	.27
S.E. ±	110	.67	.50	.15	.07	.12	.13	.39	.25	.50	.05	.10	.04	.09	.25	.05	.09
—	P(t)	< .02															

PLASMA PHOSPHOLIPIDS OF NORMAL SUBJECTS

Subject	Total Lipid Phosphorus	Cardiolipin	Phosphatidic Acid	Phosphatidyl Glycerol	Sphingomyelin	Lecithin	Phosphatidyl Ethanolamine	Phosphatidyl Serine	Phosphatidyl Inositide	Cyclic Glycero-phosphoric Acid	Inorganic Phosphate	x 2	Alkali Labile Unknowns	Choline Plasmalogen	Ethanolamine Plasmalogen	Serine Plasmalogen	Unknown Plasmalogen	Acid Labile Unknowns	Alkyl Ethers	Cortisol µg/100 ml	Corticosterone µg/100 ml
	µM Phospholipid P/liter plasma																				
UL 1	2366	38.1	14.0	28.6	247.3	1634	50.2	33.8	114.0	78.6	13.3	27.7	41.6	42.4	25.6	11.1	2.8	2.8	85.2	18.3	1.60
JO 2	2109	31.6	14.1	22.8	207.9	1612	29.9	8.0	38.2	47.7	5.7	8.7	7.0	37.5	28.3	11.8	1.3	2.7	95.3	14.9	.62
WI 3	2638	41.7	26.1	24.5	315.3	1923	53.8	18.2	96.3	56.5	12.1	4.5	26.7	39.0	19.5	4.0	3.2	10.6	99.2	10.1	.19
WIN 4	1945	16.7	19.5	14.0	293.1	1354	25.1	16.1	61.3	54.5	19.5	17.5	16.9	33.8	9.9	3.3	2.7	0	81.7	10.7	.24
HA 5	1768	30.9	10.8	19.6	306.2	1053	58.7	25.5	66.6	74.1	17.0	20.9	20.0	29.9	15.7	6.7	10.8	24.2	56.0	10.9	.28
RO 6	1556	20.9	12.0	21.3	239.5	1031	19.5	9.8	143.3	28.6	7.2	8.7	17.9	28.0	4.7	4.7	0	0	58.7	10.9	.35
YO 7	2839	29.8	17.3	19.9	372.2	2045	51.4	20.7	81.2	68.4	9.7	11.9	22.2	48.0	10.8	6.8	4.5	16.2	144.0	10.5	.64
KI 8	1962	36.5	13.5	15.7	273.2	1371	18.2	20.4	72.8	38.8	16.9	11.8	10.0	33.4	12.6	8.8	3.1	15.7	87.1	21.0	1.80
BL 9	1768	24.9	19.6	13.4	246.9	1237	28.6	13.6	64.2	45.1	12.0	12.0	8.1	22.1	17.7	7.4	5.3	17.7	60.5	16.0	.36
BR 10	1998	22.4	10.4	18.8	313.5	1401	24.6	21.6	56.3	48.4	9.4	6.0	5.4	41.8	17.6	11.4	10.6	17.2	56.9	11.0	.46
MA 11	1639	31.5	15.1	19.5	205.6	1084	26.4	16.4	90.3	49.8	14.8	9.3	17.4	31.6	19.5	7.7	9.2	15.6	60.8	12.1	.41
FR 12	2990	27.5	9.0	42.5	321.1	2314	52.9	20.9	83.4	92.7	6.6	15.9	12.6	41.3	19.7	14.1	11.7	10.8	48.1	12.4	.39
JA 13	1903	32.5	8.0	41.1	224.6	1255	53.7	21.3	78.2	95.6	5.1	19.4	28.6	44.9	24.0	17.1	12.9	15.0	20.6	10.2	.58
EV 14	2198	43.5	16.7	38.7	216.7	1394	50.8	20.2	182.6	133.8	20.2	10.8	17.8	56.3	23.7	16.0	10.6	15.4	53.0	10.8	.82
DP 15	2356	22.9	8.3	36.5	232.3	1682	42.9	16.3	114.1	92.8	9.0	16.3	24.3	53.7	24.7	18.4	14.9	18.4	56.8	11.7	.40
WID 16	1824	20.8	12.0	40.5	151.4	1237	37.8	15.0	93.9	82.1	6.4	19.9	22.3	39.6	15.5	19.7	6.9	25.7	74.6	11.6	.43
PA 17	2109	32.9	15.6	32.7	304.7	1364	40.5	17.9	104.2	98.1	8.0	15.6	20.2	62.6	17.5	6.5	4.0	16.7	53.6	12.4	.28
OL 18	2588	33.6	10.9	21.5	390.2	1772	53.3	27.4	82.3	106.6	20.2	20.2	27.4	44.5	16.3	6.5	13.7	17.6	47.9	23.1	1.05
MH 19	1824	21.5	12.8	30.3	246.0	1144	30.3	11.3	114.4	90.8	17.5	22.3	22.3	42.0	26.8	13.0	18.1	16.6	35.9	14.7	.50
JON 20	2019	18.4	11.5	27.3	269.5	1410	28.5	14.9	60.0	85.6	7.5	14.9	20.0	42.6	11.7	13.5	6.3	20.0	54.3	7.4	.41
MI 21	3247	44.2	6.8	19.2	270.2	2474	55.2	10.7	243.2	174.1	12.7	13.6	6.3	25.3	6.8	4.9	14.9	29.2	84.4	10.2	.76
CO 22	2163	30.3	6.5	8.7	244.4	1508	45.1	4.3	69.6	93.0	4.3	8.7	9.9	36.8	6.5	6.5	4.3	30.0	164.4	12.6	.82
PAT 23	2393	32.1	16.3	12.2	248.7	1872	39.0	10.1	50.6	56.6	2.9	6.0	5.1	53.6	1.0	7.9	7.2	29.7	68.5	26.7	2.59
DO 24	2918	35.0	8.8	55.4	417.3	2001	62.4	35.0	69.6	93.4	5.8	29.2	7.6	40.9	5.8	2.9	15.8	11.7	189.7	23.1	1.35
KA 25	1662	29.8	11.3	22.9	207.8	1051	47.0	14.0	79.0	49.4	10.1	10.1	4.8	34.4	19.9	21.4	17.1	38.6	80.3	6.9	.38
JE 26	2520	37.1	21.4	24.7	232.9	1784	34.8	24.7	92.5	101.6	17.1	11.6	24.7	47.1	24.2	22.7	18.7	64.3	89.7	7.1	.19
SW 27	2140	33.2	25.5	29.7	160.1	1407	28.7	22.0	109.6	142.7	24.4	29.7	16.7	37.9	33.8	20.3	41.5	12.0	93.5	9.9	.60
PAG 28	2175	33.9	15.4	17.0	253.6	1512	27.6	16.3	86.8	68.1	23.9	17.0	9.2	50.5	24.1	20.2	20.9	25.7	71.5	9.3	.23
MO 29	2550	22.4	10.6	4.1	516.4	1512	35.7	2.0	57.9	24.5	3.3	7.4	0	42.6	0	6.7	2.8	35.1	329.5	16.2	1.03
PO 30	3309	29.4	10.6	21.2	645.5	2143	26.8	5.3	76.8	45.3	2.6	5.3	0	46.3	9	7.9	4.3	93.3	177.7	13.1	.87
FI 31	2499	25.8	18.3	18.3	142.0	1584	64.6	15.1	34.6	154.9	24.8	9.0	36.6	40.9	31.2	9.7	14.0	14.0	113.0		
FII 32	2733	31.2	19.4	26.9	111.9	1894	67.8	19.4	111.4	235.6	20.4	31.3	61.2	36.6	42.0	10.7	17.2	10.7	105.4		
Mean	2272	30.1	14.2	24.7	275.9	1564	41.0	17.2	90.0	84.8	12.4	14.8	17.8	40.2	17.4	11.1	10.4	22.9	90.6	13.2	.69
S.D. ±	466	7.0	5.0	10.9	105.9	375	14.6	7.6	40.9	45.2	7.1	7.4	12.8	8.9	9.1	6.2	6.2	22.3	56.7	4.9	.54
S.E. ±	84	1.3	.9	2.0	18.7	67	2.5	1.4	7.3	8.1	1.3	1.3	2.3	1.6	1.6	1.1	1.5	4.0	10.5	.9	.1

EFFECTS OF ACCELERATION STRESS ON PLASMA PHOSPHOLIPIDS

	Total Lipid Phosphorus		Cardiolipin		Phosphatidic Acid		Phosphatidyl Glycerol		Sphingomyelin		Lecithin		Phosphatidyl Ethanolamine		Phosphatidyl Serine		Phosphatidyl Inositide		Cyclic Glycero-phosphoric Acid		Inorganic Phosphate	
	μM Phospholipid P/liter plasma																					
	Before	After	Before	After	Before	After	Before	After	Before	After	Before	After	Before	After	Before	After	Before	After	Before	After	Before	After
UL 1	2366	2346	38.1	50.4	14.0	15.5	28.6	74.8	247.3	269.8	1634	1466	50.2	89.6	33.8	39.7	114.0	151.6	78.6	102.8	13.3	15.5
JO 2	2109	1881	31.6	31.8	14.1	13.7	22.8	48.2	207.9	245.7	1612	1321	29.9	27.8	8.0	17.5	38.2	44.6	47.7	42.1	5.7	7.7
WI 3	2638	2535	41.7	55.3	26.1	20.0	24.5	49.2	315.3	341.7	1923	1657	53.8	102.2	18.2	24.3	96.3	56.8	56.5	82.4	12.1	26.6
WIN 4	1945	1989	16.7	25.9	19.5	15.5	14.0	29.2	293.1	344.3	1354	1317	25.1	33.6	16.1	18.5	61.3	59.3	54.5	63.5	19.5	8.6
HA 5	1768	1768	30.9	23.2	10.8	17.3	19.6	45.8	306.2	296.4	1053	1081	58.7	65.1	25.5	10.4	66.6	48.8	74.1	42.1	17.0	23.5
RO 6	1556	1707	20.9	20.0	12.0	24.8	21.3	32.1	239.5	226.0	1031	1148	19.5	44.2	9.8	10.1	143.3	39.6	28.6	58.4	7.2	3.4
YO 7	2839	2405	29.8	38.5	17.3	21.9	19.9	41.1	372.2	307.3	2045	1613	51.4	42.1	20.7	22.1	81.2	110.9	68.4	85.4	9.7	17.6
KI 8	1962	2044	36.5	22.1	13.5	18.4	15.7	36.6	273.2	329.4	1371	1233	18.2	72.1	20.4	24.1	72.8	89.1	38.8	94.4	16.9	15.7
BL 9	1768	2081	24.9	17.1	19.6	14.4	13.4	34.6	246.9	292.0	1237	1394	28.6	61.4	13.6	14.4	64.2	87.0	45.1	63.5	12.0	10.8
BR 10	1998	2118	22.4	21.8	10.4	11.2	18.8	29.7	313.5	298.5	1401	1552	24.6	38.3	21.6	15.3	56.3	47.2	48.4	43.8	9.4	5.1
MA 11	1639	2268	31.5	29.0	15.1	15.2	19.5	39.7	205.0	331.6	1084	1543	26.4	34.7	18.4	16.3	90.3	90.1	49.8	83.3	14.8	12.3
FR 12	2990	2625	27.5	20.7	9.0	11.0	42.5	54.3	321.1	300.1	2314	1883	52.9	54.3	20.9	20.5	83.4	112.9	92.7	90.8	6.6	18.4
JA 13	1903	2429	32.5	20.2	8.0	28.7	41.1	57.8	224.6	230.8	1255	1666	53.7	67.5	21.3	32.8	78.2	121.0	95.0	127.0	5.1	12.6
EV 14	2198	2120	43.5	23.5	16.7	21.2	38.7	75.5	216.7	230.4	1394	1430	50.8	57.2	20.2	15.1	182.6	82.0	133.8	68.0	26.2	9.8
DP 15	2356	1929	22.9	39.0	8.3	33.4	36.5	68.5	232.3	242.2	1682	1169	42.9	58.4	16.3	21.6	114.1	124.8	92.8	105.1	9.0	8.3
WID 16	1824	2000	20.8	19.4	12.0	10.4	40.5	53.2	151.4	261.6	1237	1287	37.8	44.2	15.0	11.2	93.9	92.6	82.1	121.0	6.4	19.0
PA 17	2109	1764	32.9	15.7	15.6	26.3	32.7	54.2	304.7	268.3	1364	1097	40.5	37.2	17.9	12.7	104.2	70.6	98.1	81.1	8.0	4.1
OL 18	2588	2752	33.6	25.1	10.9	23.7	21.5	56.7	390.2	417.8	1772	1911	53.3	59.2	27.4	22.6	82.3	54.0	106.6	70.5	20.2	19.3
MH 19	1824	1981	21.5	14.7	12.8	17.2	30.3	44.7	246.0	292.0	1144	1292	30.3	39.4	11.3	18.8	114.4	100.2	96.8	57.8	17.5	16.8
JON 20	2019	2190	18.4	21.5	11.5	22.3	27.3	63.1	269.5	325.0	1410	1271	26.5	67.0	14.9	24.1	60.0	135.1	85.0	115.0	7.5	16.4
MI 21	3247	3476	44.2	21.6	6.8	12.5	19.2	80.7	270.2	538.2	2474	2416	55.2	48.0	10.7	9.7	243.2	91.4	174.1	184.3	12.7	11.5
CO 22	2163	2240	30.3	11.2	6.5	11.2	8.7	58.2	244.4	362.9	1508	1558	45.1	51.5	4.3	6.7	69.6	50.1	93.0	24.6	4.3	11.2
PAT 23	2393	2652	32.1	10.6	16.3	19.4	12.2	57.0	248.7	476.7	1872	1796	39.0	61.0	10.1	6.9	50.6	72.6	56.0	76.1	2.9	5.0
DO 24	2918	3194	35.0	16.0	8.8	12.8	55.4	95.8	417.3	610.1	2001	2062	62.4	44.7	35.0	41.5	69.6	62.7	93.4	165.8	5.8	12.8
Mean	2213	2271	30.0	24.8	13.2	18.3	26.0	53.4	273.2	326.6	1549	1507	40.8	54.2	18.0	19.0	92.9	83.1	78.7	85.4	11.2	13.0
S.E. ±	95	91	1.6	2.3	1.0	1.3	2.4	3.5	12.9	20.2	82.2	68.6	2.8	3.7	1.6	1.9	9.4	6.5	6.8	7.9	1.2	1.3

EFFECTS OF ACCELERATION STRESS ON PLASMA PHOSPHOLIPIDS

	%		Alkali Labile Unknowns		Choline Plasmalogen		Ethanolamine Plasmalogen		Serine Plasmalogen		Unknown Plasmalogen		Acid Labile Unknowns		Alkyl Ethers		Cortisol µg/100 ml		Corticosterone µg/100 ml	
	µM Phospholipid P/liter plasma																			
	Before	After	Before	After	Before	After	Before	After	Before	After	Before	After	Before	After	Before	After	Before	After	Before	After
UL 1	27.7	7.7	41.6	21.8	42.4	36.4	25.6	22.5	11.1	2.8	2.8	0	2.8	6.1	85.2	99.2	18.3	21.8	1.60	.84
JO 2	8.7	18.3	7.0	24.3	37.5	20.1	28.3	5.5	11.8	0	1.3	0	2.7	.8	95.3	99.7	14.9	21.6	.62	1.36
WI 3	4.5	3.6	26.7	17.5	39.0	52.2	19.5	23.8	4.0	7.1	3.2	2.0	10.6	10.4	99.2	112.8	10.1	9.6	.19	.12
WIN 4	17.5	11.1	16.9	8.4	33.8	43.2	9.9	9.8	3.3	5.4	2.7	0	0	0	81.7	87.5	10.7	19.4	.24	.29
HA 5	20.9	14.0	20.0	31.8	29.9	37.1	15.7	16.1	6.7	8.8	10.8	3.0	24.2	21.4	56.0	56.7	10.9	12.2	.28	.79
RO 6	8.7	5.0	17.9	27.3	28.0	30.6	4.7	26.6	4.7	12.6	0	7.5	0	1.2	58.7	66.2	10.9	17.7	.35	1.31
YO 7	11.9	29.6	22.2	20.7	48.0	39.4	10.8	9.1	6.8	4.3	4.5	5.8	16.2	11.3	144.0	107.5	10.5	7.6	.64	.76
KI 8	11.8	21.3	10.0	23.1	33.4	37.6	12.6	9.4	8.8	5.9	3.1	0	15.7	15.1	87.1	91.6	21.0	21.2	1.80	1.70
BL 9	12.0	22.1	8.1	20.4	22.1	41.6	17.7	12.9	7.4	8.7	5.3	9.4	17.7	14.8	60.5	66.0	16.0	15.0	.36	.63
BR 10	6.0	5.1	5.4	8.7	41.8	39.2	17.6	22.0	11.4	9.3	10.6	10.8	17.2	8.1	56.9	53.4	11.0	19.2	.46	1.51
MA 11	9.3	22.5	17.4	20.9	31.6	39.0	19.5	18.8	7.7	5.9	9.2	9.3	15.6	14.7	60.8	54.7	12.1	17.4	.41	.75
FR 12	15.9	26.3	12.6	20.7	41.3	51.2	19.7	23.6	14.1	14.7	11.7	12.9	10.8	11.6	48.1	37.3	12.4	9.7	.39	.51
JA 13	19.4	22.1	28.6	24.5	44.9	42.8	24.0	18.5	17.1	13.4	12.9	10.0	15.0	16.3	20.6	46.9	10.2	8.5	.58	.25
EV 14	10.8	15.3	17.8	12.7	56.3	54.9	23.7	27.6	18.0	20.1	10.6	16.0	15.4	11.0	53.0	51.7	10.8	11.5	.32	.83
DP 15	16.3	30.1	24.3	15.8	53.7	52.5	24.7	10.0	18.4	6.8	14.9	1.7	18.4	14.1	56.8	28.9	11.7	12.1	.40	.39
WID 16	19.9	16.8	22.3	2.8	39.6	44.4	15.5	22.8	19.7	9.0	6.9	6.6	25.7	16.4	74.6	60.8	11.6	15.6	.43	.49
PA 17	15.6	16.2	20.2	9.0	62.6	44.6	17.5	19.4	6.5	13.2	4.0	15.2	16.7	21.5	53.6	39.7	12.4	12.0	.28	.52
OL 18	20.2	25.1	27.4	16.8	44.5	44.9	16.3	22.6	6.5	19.3	13.7	18.7	17.6	16.8	47.9	51.8	23.1	12.1	1.05	.38
MH 19	22.3	18.8	22.3	19.6	42.0	37.8	26.8	14.1	13.0	19.6	18.1	11.1	16.6	19.6	35.9	47.7	14.7	21.8	.50	2.03
JON 20	14.9	28.5	20.0	25.4	42.6	45.8	11.7	22.8	13.5	12.9	6.3	9.2	20.0	19.9	54.3	75.3	7.4	8.0	.41	.45
MI 21	13.6	13.2	6.3	9.6	25.3	48.3	6.8	16.3	4.9	12.2	14.9	14.3	29.2	35.8	84.4	93.2	10.2	20.6	.76	2.80
CO 22	8.7	15.7	9.9	5.1	36.8	24.6	6.5	6.7	6.5	2.2	4.3	4.5	30.0	17.9	164.4	127.7	12.6	26.8	.82	1.47
PAT 23	6.0	25.7	5.1	19.5	53.6	57.3	1.0	8.8	7.9	7.7	7.2	14.9	29.7	6.1	68.5	79.0	26.7	14.3	2.59	.53
DO 24	29.2	22.4	7.6	12.1	40.9	41.5	5.8	9.6	2.9	3.2	15.8	0	11.7	9.9	189.7	83.0	23.1	20.8	1.35	2.23
Mean	14.7	18.2	17.4	17.4	40.5	42.0	15.9	16.6	9.7	9.4	8.1	7.7	15.8	13.4	76.6	71.6	13.9	15.7	.72	.95
S.E. ±	1.4	1.6	1.8	1.5	2.0	1.9	1.6	1.4	1.0	1.2	1.1	1.3	1.8	1.6	8.3	5.5	1.0	1.1	.12	.15

CONTROL ACCELERATION STRESS AND RECOVERY PATTERNS OF HUMAN PLASMA PHOSPHOLIPIDS

	Total Lipid Phosphorus	Lecithin	Phosphatidyl Ethanolamine	Phosphatidyl Serine	Cardiolipin	Phosphatidic Acid	Phosphatidyl Glycerol	Phosphatidyl Inositide	Cyclic Glycero-phosphoric Acid	X_2	Inorganic Phosphate	Choline Plasmalogen	Ethanolamine Plasmalogen	Serine Plasmalogen	Unknown Plasmalogen	Sphingomyelin	Acid Labile Unknown	Alkyl Ethers	Cortisol µg/100 ml	Corticosterone µg/100 ml		
	µM Phospholipid P/liter plasma																					
Before Acceleration																						
MI 1	3247	2474	55.2	10.7	44.2	6.8	19.2	243.2	174.1	13.6	12.7	25.3	6.8	4.9	14.9	270.2	29.2	84.4	10.2	.76		
CO 2	2163	1509	45.1	4.3	30.3	6.5	8.7	69.6	93.0	8.7	4.3	36.8	6.5	6.5	4.3	244.4	30.0	164.4	12.6	.82		
OL 3	2557	1700	59.8	15.6	38.7	12.0	38.6	74.1	132.2	16.4	26.6	38.6	18.2	5.1	12.3	342.8	17.6	103.5	16.6	1.01		
PA 4	2394	1872	39.0	10.1	32.1	16.3	12.2	50.6	56.0	6.0	2.9	53.6	1.0	7.9	7.2	248.7	29.7	68.5	26.7	2.59		
DO 5	2918	2009	63.3	35.9	35.9	9.6	55.2	67.1	93.1	29.8	5.8	40.6	6.1	2.3	5.3	419.0	11.7	161.1	23.1	1.35		
PAQ 6	2199	1418	68.3	28.6	39.6	22.0	30.8	81.4	61.6	22.0	24.2	15.4	50.6	26.4	17.6	217.7	13.2	162.7				
Mean	2580	1830	55.1	17.5	36.8	12.2	27.5	97.7	101.7	16.1	12.8	35.1	14.9	8.9	10.3	290.5	21.9	124.1	17.8	1.31		
S.E. ±	175	157	4.6	5.0	2.1	2.5	7.2	29.4	18.3	3.6	4.2	5.4	7.5	3.6	2.2	31.0	3.5	17.9	3.1	.34		
Immediately After Acceleration																						
MI 1	3476	2416	48.0	9.7	21.6	12.5	80.7	91.4	184.3	13.2	11.5	48.3	16.3	12.2	14.3	538.2	35.8	93.2	20.6	2.80		
CO 2	2240	1558	51.5	6.7	11.2	11.2	58.2	50.1	24.6	15.7	11.2	24.6	6.7	2.2	4.5	362.9	17.9	127.7	26.8	1.47		
OL 3	2505	1750	53.4	19.3	18.5	23.8	47.3	87.3	120.7	20.8	45.8	67.9	16.3	7.3	11.3	309.1	9.8	22.5	16.0	.82		
PA 4	2652	1796	61.0	6.9	10.6	19.4	57.0	72.6	76.1	25.7	5.0	57.3	8.8	7.7	14.9	475.7	6.1	79.0	14.3	.53		
DO 5	3194	2062	44.7	41.5	16.0	12.8	95.8	62.7	165.8	22.4	12.8	41.5	9.6	3.2	0	629.5	9.9	83.0	20.8	2.23		
PAQ 6	2168	1264	78.1	23.8	30.4	52.0	69.4	117.1	75.9	26.0	28.2	34.7	21.7	30.4	13.0	260.2	21.7	125.7				
Mean	2706	1807	56.1	18.0	18.1	22.0	68.1	80.2	107.9	20.6	19.1	45.7	13.2	10.5	9.7	429.3	16.9	88.5	19.7	1.57		
S.E. ±	215	163	4.9	5.5	3.0	6.3	7.3	9.7	24.7	2.1	6.2	6.4	2.4	4.2	2.5	58.2	4.5	15.7	2.2	.42		
3 Hours After Acceleration																						
MI 1	3056	2178	61.1	21.4	16.8	25.7	87.4	105.1	167.2	6.4	37.9	32.1	9.8	4.3	11.6	364.6	12.5	66.9	11.0	1.12		
CO 2	2095	1199	113.2	33.5	35.6	37.7	52.4	102.7	71.2	25.1	23.0	39.8	25.1	12.6	6.3	276.6	14.7	100.6	10.4	.62		
OL 3	2486	1706	54.7	18.6	17.4	17.9	45.2	58.2	64.4	8.7	8.2	42.3	4.5	7.7	14.4	478.4	8.7	38.3	11.5	2.20		
PA 4	2666	1776	47.2	9.0	14.9	5.6		58.4	108.0	11.2	10.2	22.4	4.2	5.8	17.0	515.6	1.3	36.2	18.5	1.54		
DO 5	2871	1981	61.4	11.2	13.2	11.2	53.4	84.0	122.3	8.0	10.0	46.5	9.2	3.4	5.5	500.5	25.3	62.0	5.9	.44		
PAQ 6	2233	1534	46.9	22.3	20.1	29.0	20.1	89.3	46.9	22.3	17.9	33.5	20.1	15.6	13.4	225.4	17.9	147.4				
Mean	2568	1729	64.1	19.3	19.7	21.2	51.7	83.0	96.7	13.6	17.9	36.1	12.2	8.2	11.4	393.5	13.4	75.2	11.5	1.18		
S.E. ±	151	140	10.2	3.6	3.3	4.9	10.8	8.4	18.2	3.3	4.6	3.5	3.5	2.0	1.9	50.4	3.3	17.3	2.0	.32		
Recovery - 24 Hours After Acceleration																						
MI 1	3109	2291	90.8	47.2	31.7	6.8	26.1	109.2	166.6	7.8	11.2	50.4	3.7	5.0	9.6	340.1	14.6	57.2	9.7	1.75		
CO 2	2211	1559	75.2	28.7	31.0	11.1	11.1	52.6	68.5	19.9	8.8	53.1	6.6	6.6	4.4	309.6	19.9	50.9	8.4	.53		
OL 3	2550	1832	35.2	5.1	9.4	7.9	10.2	110.4	102.8	15.3	8.7	44.1	5.4	8.4	8.7	435.9	5.6	23.2	11.2	.83		
PA 4	2384	1894	27.7	3.8	21.9	6.7	11.2	71.1	71.8	16.0	13.1	37.4	6.0	4.3	10.3	270.1	10.3	35.5	9.7	1.25		
DO 5	2773	1894	39.4	10.3	35.5	11.1	15.0	95.6	89.3	7.2	12.2	25.5	8.0	3.9	0	603.9	19.4	36.0	9.3	1.27		
PAQ 6	2296	1474	57.4	41.3	43.6	18.4	29.8	137.8	78.1	16.1	27.6	0	43.6	27.6	29.8	248.0	27.6	121.7				
Mean	2554	1824	54.3	22.7	28.9	10.3	17.2	96.1	96.2	13.7	13.6	35.1	12.2	9.3	10.5	367.9	16.2	54.1	9.7	1.13		
S.E. ±	138	118	10.1	7.8	4.8	1.8	3.5	12.4	15.0	2.1	2.9	8.1	6.3	3.7	4.2	54.3	3.2	14.4	.5	.21		

PLASMA PHOSPHOLIPIDS OF CHRONIC SCHIZOPHRENIC PATIENTS

Subject	Total Lipid Phosphorus	Lecithin	Cardiolipin	Phosphatidic Acid	Phosphatidyl Glycerol	Sphingomyelin	Phosphatidyl Ethanolamine	Phosphatidyl Serine	Phosphatidyl Inositide	Cyclic Glycero-phosphoric Acid	X_2	Inorganic Phosphate	Choline Plasmalogen	Ethanolamine Plasmalogen	Serine Plasmalogen	Unknown Plasmalogen	Alkyl Ethers	Alkali Labile Unknowns	Acid Labile Unknowns	Cortisol ug/100 ml	Corticosterone ug/100 ml
	μM Phospholipid P/liter plasma																				
JF	2574	1888	23.2	41.2	41.2	285.7	121.0	28.3	122.5	64.3	22.7	23.4	36.0	23.2	12.9	10.3		.4			
MR	3147	2302	25.2	44.1	47.2	251.8	138.4	31.5	158.6	78.7	36.8	20.2	28.3	18.9	6.3	12.6		52.9			
EW	3530	2791	28.2	45.9	49.4	321.2	67.1	52.9	148.3	74.1	30.0	18.2	28.2	21.2	10.6	7.1		69.2			
TL	2973	2051	50.5	56.5	98.1	282.4	101.1	41.6	166.5	142.7	34.2	29.0	32.7	26.8	11.9	17.9		56.3			
MS	2574	1346	62.4	66.7	61.3	298.0	116.3	51.6	165.7	293.7	42.5	40.4	30.1	35.5	26.9	30.1	129.1	66.7	30.1	16.9	.81
CH	2306	1525	45.2	21.6	38.7	113.0	62.4	34.4	82.9	184.0	18.2	34.4	37.7	55.9	17.2	44.1	74.2	28.0	44.0	18.5	.88
JK	2301	1437	59.2	57.0	63.5	267.9	104.4	23.7	212.4	264.0	36.6	46.3	64.6	20.0	31.2	36.7	100.1	65.6	38.7	11.9	.86
SW	2294	1405	60.2	47.3	43.0	192.6	94.7	36.6	167.2	164.6	27.3	18.3	91.4	21.5	31.2	30.1	189.3	46.3	30.0	12.2	.62
WM	2807	1756	40.7	25.5	63.4	492.7	78.6	18.2	55.0	74.1	31.4	13.5	84.8	31.2	15.4	20.5	87.6	8.1	25.0	7.2	.32
TB	2576	1556	46.6	24.7	63.4	416.1	77.5	34.5	89.9	113.1	16.7	15.7	53.3	17.0	16.2	12.9	94.3	18.6	22.9	11.6	.60
SB	2841	1709	45.6	41.5	75.6	530.9	87.8	30.4	52.8	128.1	27.0	10.2	61.4	29.0	12.8	13.9	50.3	27.0	16.2	13.9	.71
HK	2253	1478	18.7	20.3	43.9	451.2	29.5	11.5	44.2	73.4	17.8	14.9	55.0	7.7	4.5	12.6	42.6	12.4	10.4	11.9	.34
AC	2340	1620	26.6	11.9	31.4	355.4	45.1	16.4	65.4	78.6	5.6	18.7	32.1	11.2	13.3	25.3	37.4	11.9	17.1	8.6	.65
RR	2951	2171	60.5	45.4	81.4	199.5	92.4	26.9	86.8	138.7	40.4	13.6	23.3	9.7	14.5	13.9	44.9	24.2	11.8	8.9	.46
AR	2543	1841	22.4	10.9	48.1	157.4	92.6	20.6	126.7	59.0	24.2	17.3	63.1	59.3	16.5	13.5	46.5	17.0	47.6	8.9	1.52
JS	2346	1476	23.0	15.7	54.4	377.0	57.9	20.6	68.0	64.3	16.7	0	87.7	23.0	21.3	17.6	63.3	11.7	31.2	8.9	1.37
EW	2475	1723	21.5	24.3	33.9	264.8	50.2	31.2	81.4	75.2	20.5	21.5	62.4	14.8	18.1	18.6	46.0	25.2	42.6	17.3	.82
JK	2807	1806	56.1	47.4	71.6	261.0	117.6	51.9	270.0	76.6	13.2	10.4	55.0	18.5	10.9	5.9	62.9	14.6	35.6	10.7	.52
MS	2586	1618	46.8	39.6	59.0	332.6	67.0	33.4	69.8	122.1	22.2	17.1	80.0	20.7	18.9	18.1	65.2	34.1	33.6	14.0	.83
SW	2193	1381	34.2	26.3	66.4	252.2	61.2	27.0	84.6	91.9	20.2	16.0	90.1	20.2	20.2	12.9	49.6	22.6	16.4	10.0	.38
Mean	2621	1744	40.1	35.7	56.8	306.2	83.2	31.2	117.9	119.1	25.2	20.4	54.9	24.7	16.5	18.8	75.2	30.6	81.2	12.0	.73
S.D. ±	346	361	15.3	16.1	17.0	107.9	28.6	11.7	59.4	68.3	9.7	11.6	23.1	13.4	7.1	10.1	39.8	21.3	28.2	3.4	.34
S.E. ±	79	83	3.5	3.7	3.9	24.8	6.6	2.7	13.6	15.7	2.2	2.6	5.3	3.1	1.6	2.3	9.1	4.8	7.0	.9	.09

SLEEP DEPRIVATION STRESS AND RECOVERY PATTERNS OF HUMAN PLASMA PHOSPHOLIPIDS

	Total Lipid Phosphorus	Lecithin	Phosphatidyl Ethanolamine	Phosphatidyl Serine	Cardiolipin	Phosphatidic Acid	Phosphatidyl Glycerol	Phosphatidyl Inositide	Cyclic Glycero-phosphoric Acid	Inorganic Phosphorus	X_2	Choline Plasmalogen	Ethanolamine Plasmalogen	Serine Plasmalogen	Sphingomyelin	Alkyl Ethers	Cortisol µg/100 ml	Corticosterone µg/100 ml
	µM Phospholipid P/liter Plasma																	
Before Sleep Deprivation																		
Ka	1662	1051	47.0	14.0	29.8	11.3	22.9	79.0	49.4	10.1	10.1	34.4	19.9	21.4	207.8	80.3	6.87	.38
Co	1792	1179	31.0	24.9	31.0	15.8	23.8	75.2	53.6	23.8	18.8	57.3	17.2	24.2	195.3	60.7	7.37	1.03
Je	2520	1784	34.6	24.7	37.1	21.4	24.7	92.5	101.6	17.1	11.6	47.1	24.2	22.7	232.9	89.7	7.13	.19
Cr	2711	1895	46.7	10.8	35.2	21.7	29.8	62.4	124.7	5.4	19.6	100.3	13.6	8.1	338.9	97.6	9.24	.53
Do	2430	1626	72.9	26.7	29.2	19.4	17.0	89.9	136.5	19.4	26.7							.33
Sw	2140	1407	26.7	22.0	33.2	25.5	29.7	109.6	142.7	24.4	29.7	37.9	33.8	20.3	160.1	93.5	9.87	.60
Pa	2175	1512	27.6	16.3	33.9	15.4	17.0	80.6	68.1	23.9	17.0	50.5	24.1	20.2	253.6	71.5	9.25	.23
Mean	2204	1493	40.4	19.9	32.8	18.6	23.6	95.1	96.9	17.7	19.0	54.6	22.1	19.5	231.4	82.2	8.29	.47
S.E. ±	145	116	6.0	2.3	1.1	1.8	2.6	5.6	15.1	2.8	2.7	9.7	2.9	2.3	25.2	5.8	.53	.11
After Sleep Deprivation																		
Ka	1588	918	24.5	16.9	21.8	26.3	29.1	69.9	51.6	28.3	24.5	45.3	12.5	21.3	219.0	85.0	5.92	.27
Co	1770	1170	30.1	22.1	24.4	39.5	35.9	66.9	51.2	16.5	22.1	36.3	19.8	18.4	201.2	55.0	5.29	1.05
Je	2330	1645	40.5	18.4	24.9	31.2	34.2	80.6	70.6	11.4	14.2	31.2	16.5	22.6	262.1	82.5	5.63	.23
Cr	2782	1878	47.3	25.0	33.4	30.6	50.1	64.0	83.5	5.6	8.3	108.5	13.9	11.1	344.9	158.6	9.33	.42
Do	2272	1413	95.4	34.1	22.7	43.2	47.7	140.8	88.6	22.7	31.8						6.30	.91
Sw	1870	1204	44.9	24.1	23.0	36.5	39.8	91.8	47.1	32.3	23.0	57.6	42.8	11.8	167.5	72.0	8.50	.56
Pa	2305	1579	45.2	18.7	26.5	35.7	47.3	104.4	69.4	21.4	18.7	37.8	30.4	29.5	220.4	80.9	5.31	.69
Mean	2131	1401	46.8	23.0	25.2	35.0	40.6	88.3	66.0	19.7	20.4	52.8	22.7	19.1	235.9	89.0	6.64	.59
S.E. ±	155	124	8.7	2.1	1.5	2.0	3.0	10.3	6.2	3.5	2.9	11.8	4.8	2.9	25.1	14.6	.61	.12
Recovery																		
Ka	1636	951	92.7	25.8	39.1	17.0	25.8	48.1	117.8	20.4	12.6	39.9	15.9	18.5	136.4	103.5	14.33	.75
Co	1769	1292	60.0	11.7	29.4	14.7	16.6	64.4	61.6	14.7	16.8	25.1	13.4	15.0	139.8	52.9	7.32	.17
Je	2171	1344	44.9	17.8	33.2	18.7	21.5	85.5	84.4	11.1	20.6	47.5	18.2	16.9	305.9	93.3	4.23	.17
Cr	2490	1688	79.7	24.9	37.4	19.9	24.9	137.0	127.0	37.4	19.9	57.3	7.5	5.0	214.2	112.1	14.52	1.20
Do	2292	1549	57.3	34.4	27.5	27.5	18.3	100.8	94.0	27.5	18.3						6.99	1.33
Sw	2171	1410	29.3	20.2	39.9	25.4	24.3	121.1	69.0	29.3	20.2	63.8	36.0	7.8	234.4	112.9	10.11	.53
Pa	2260	1471	26.7	16.1	32.5	21.7	26.7	59.4	75.5	18.8	20.8	49.3	26.7	24.0	326.5	112.1	16.28	.91
Mean	2113	1387	55.8	21.9	34.1	20.7	22.6	88.0	89.9	22.7	18.5	47.2	19.6	14.5	226.2	97.8	10.54	.72
S.E. ±	114	88	9.3	2.7	1.8	1.7	1.5	12.6	9.3	3.3	1.1	5.5	4.2	2.9	32.7	9.5	1.73	.17

PLASMA PHOSPHOLIPID LEVELS AFTER BEGINNING COMBAT FLIGHTS

		Total Lipid Phosphorus	Cardiolipin	Phosphatidic Acid	Phosphatidyl Glycerol	Sphingomyelin	Lecithin	Phosphatidyl Ethanolamine	Phosphatidyl Serine	Phosphatidyl Inositide	Cyclic Glycero-phosphoric Acid	Choline Plasmalogen	Ethanolamine Plasmalogen	Serine Plasmalogen	Alkyl Ethers	Inorganic Phosphorus	X_2	Alkali Labile Unknown	Unknown Plasmalogen	Acid Labile Unknown	Cortisol µg/100 ml	Time of Sampling		
		µM Phospholipid P/liter Plasma																						
KE	1	3467	46.8	21.5	55.5	447.6	2523	143.2	28.4	159.1	39.2	34.7	23.2	0	89.5	8.7	5.5	0	0	29.5	12.2	1200		
LE	2	3434	27.5	6.9	9.3	365.7	2823	64.9	19.2	50.8	91.6	52.9	18.9	0	53.2	6.9	0	0	0	17.8	16.2	1215		
MG	3	2975	22.6	7.1	14.0	439.7	2121	33.3	14.0	366.2	28.5	44.6	13.7	0	29.8	2.1	2.1	0	0	60.7	23.5	1215		
HO	4	2830	40.5	18.2	35.2	476.0	2051	42.6	20.7	49.6	60.3	56.4	0	0	41.2	11.7	33.6	0	0	23.3	-	1600		
PO	5	3548	38.7	6.0	41.1	356.2	2773	118.2	19.4	122.8	45.8	61.4	13.8	12.0	22.4	24.8	26.7	18.4	23.0	15.7	-	1730		
RE	6	3079	46.8	21.6	44.3	398.4	2097	74.5	33.9	100.4	162.2	66.8	15.1	13.9	34.2	28.9	27.1	25.2	23.7	12.3	17.7	1700		
FI	7	2329	22.1	21.4	30.5	378.8	1452	51.2	21.4	74.1	102.0	59.2	14.4	14.4	67.8	25.2	23.5	21.4	22.6	29.6	13.1	1200		
REA	8	2916	53.1	28.6	31.2	429.3	2000	74.9	25.4	91.6	81.7	22.7	2.9	0	43.2	28.6	29.7	45.2	18.7	49.3	14.2	1630		
SM	9	3186	37.3	18.8	27.0	540.6	2208	82.8	33.8	90.7	95.9	22.9	17.8	0	52.6	13.0	11.4	39.2	17.8	26.1	-	1630		
BA	10	2788	29.8	22.6	30.7	482.6	1923	49.9	13.9	99.0	83.4	46.8	16.4	15.6	29.6	17.0	9.8	13.9	32.3	9.2	12.6	2030		
MA	11	3162	29.4	9.5	47.4	377.9	2408	45.5	14.5	46.2	111.3	74.3	21.5	25.6	24.0	0	26.9	4.7	22.1	22.1	13.9	1300		
FA	12	3138	26.4	11.9	6.6	456.2	2227	52.1	31.4	74.4	132.4	83.5	56.8	27.0	15.1	3.5	6.6	6.9	51.1	15.7	19.3	1530		
DI	13	2403	30.0	13.9	24.4	492.7	1519	50.2	28.4	69.5	59.6	49.5	31.0	16.6	40.4	13.0	30.3	8.9	13.0	19.2	18.4	1900		
HY	14	2608	50.3	13.6	44.1	314.8	1852	72.8	28.7	64.9	44.6	41.7	38.9	30.3	78.5	18.3	4.7	0	0	33.1	11.0	1830		
HU	15	2917	42.6	14.3	44.0	523.9	1909	78.8	24.2	78.5	87.8	0	0	0	106.5	21.0	20.1	11.4	31.2	42.6	13.7	1600		
WE	16	2390	33.7	19.4	46.1	283.9	1641	45.2	15.8	196.2	56.6	30.6	12.0	20.6	55.2	19.4	18.2	12.4	22.2	11.0	13.0	1330		
HA	17	3672	70.9	18.7	29.4	349.9	2611	151.3	51.4	172.6	82.2	65.0	30.1	38.9	77.8	0	7.0	5.5	0	107.6	16.4	1630		
JO	18	3098	49.3	22.6	24.5	588.0	1994	70.9	37.5	68.8	130.4	41.8	25.1	18.3	53.9	7.1	15.2	22.0	22.9	37.2	13.8	2315		
MAN	19	2896	37.1	38.2	66.3	415.5	1823	86.3	34.2	290.7	78.2	49.8	32.7	23.5	37.9	11.0	13.9	11.9	18.2	13.6	21.0	1915		
FE	20	3292	46.1	14.5	27.0	542.8	2247	69.8	37.2	41.8	48.4	104.7	68.5	23.0	88.2	5.9	7.6	11.9	13.2	37.2	15.0	2300		
ST	21	3573	51.5	16.5	17.6	547.8	2380	79.7	36.1	72.6	72.6	39.3	0	0	110.6	16.5	67.6	15.0	0	115.0	25.9	2115		
OW	22	2906	32.0	26.2	28.2	543.1	1828	70.0	28.2	130.2	138.0	26.4	4.6	0	109.8	3.8	17.7	22.4	0	38.4	16.1	2330		
ZL	23	2556	32.7	21.5	71.8	351.9	1636	62.4	15.8	85.1	99.4	56.7	6.9	2.0	94.6	22.0	13.0	33.7	23.3	43.7	9.8	0015		
MI	24	2754	38.8	18.7	31.1	458.8	1812	105.5	27.0	56.2	69.1	38.0	28.6	0	62.0	17.1	15.4	43.8	0	50.4	15.2	1530		
Mean		2997	39.0	18.0	34.5	444.3	2077	74.0	26.7	110.5	83.4	48.7	20.5	11.7	59.1	13.6	18.1	15.6	14.8	35.8	15.8			
S.E.		78	2.3	1.5	3.3	18.5	76	6.1	1.9	16.1	7.0	4.5	3.5	2.5	6.0	1.8	2.9	2.8	2.8	5.5	.9			

PLASMA PHOSPHOLIPID LEVELS AFTER EXTENDED COMBAT FLIGHTS

	Total Lipid Phosphorus	Cardiolipin	Phosphatidic Acid	Phosphatidyl Glycerol	Sphingomyelin	Lecithin	Phosphatidyl Ethanolamine	Phosphatidyl Serine	Phosphatidyl Inositide	Cyclic Glycero-phosphoric Acid	Choline Plasmalogen	Ethanolamine Plasmalogen	Serine Plasmalogen	Alkyl Ethers	Inorganic Phosphate	X_2	Alkali Labile Unknown	Unknown Plasmalogen	Acid Labile Unknowns	Cortisol ug/100 ml	Time of Sampling
	μM Phospholipid P/liter plasma																				
KE 1	3817	63.5	31.4	91.7	453.6	2699	96.9	44.7	164.6	96.9	55.1	13.0	0	127.0	31.4	12.6	-	0	36.9	24.7	2000
MG 2	2981	36.9	20.5	25.0	499.2	2016	76.0	21.8	102.8	70.0	23.5	5.0	0	196.7	5.0	8.4	-	0	17.3	23.9	1515
PO 3	3589	33.7	19.1	48.5	416.4	2743	67.8	20.1	59.2	106.6	78.6	56.8	31.3	28.0	8.6	0	0	29.1	14.8	21.3	1515
RE 4	2778	34.7	31.1	55.2	389.2	1758	71.3	28.6	113.6	128.4	70.6	16.1	22.2	61.9	31.1	34.7	21.9	23.3	18.0	16.5	1330
IV 5	2567	46.5	32.0	98.0	372.2	1549	81.1	32.0	138.2	89.6	38.0	24.6	16.9	39.6	21.3	29.2	26.8	35.9	22.9	12.9	1215
FI 6	2773	43.6	36.1	86.1	487.9	1684	96.3	36.0	57.1	128.0	72.4	54.7	34.7	16.7	11.9	16.9	0	0	23.1	12.3	1200
BA 7	2760	47.2	35.6	63.7	460.4	1546	63.7	28.2	124.0	150.2	74.7	51.9	25.7	33.6	35.6	44.2	60.1	20.7	17.4	21.6	2130
MA 8	3025	29.9	21.5	44.4	580.0	1845	59.8	73.5	64.7	111.0	104.4	45.5	45.5	25.0	0	22.6	16.1	24.5	34.8	21.4	1330
FA 9	2793	14.5	13.6	63.1	517.5	1764	103.6	17.1	83.5	114.5	58.7	56.2	28.8	17.0	3.9	6.7	0	31.6	21.5	12.1	1730
WE 10	2410	26.8	31.1	51.1	228.7	1742	66.3	22.9	130.1	49.9	34.2	14.5	11.1	41.5	18.6	24.6	17.4	25.1	10.6	8.1	2215
DI 11	2400	30.9	35.5	52.8	423.4	1538	71.8	26.5	78.7	39.2	31.7	20.7	18.2	31.2	18.5	21.9	18.5	22.8	30.2	11.3	1700
IN 12	4251	62.1	20.4	62.5	764.4	2608	173.9	68.5	119.0	152.6	80.8	37.9	17.0	126.8	38.3	8.9	36.1	17.0	39.5	16.9	1530
GU 13	2782	55.1	20.9	55.6	418.9	1735	113.7	44.2	75.4	126.9	80.6	0	0	45.3	31.8	19.0	12.2	0	67.1	18.2	1930
MAN 14	2776	28.9	33.9	83.3	424.4	1828	67.5	22.2	89.7	87.7	51.6	63.6	12.5	35.5	18.3	23.9	22.2	11.1	.6	11.8	0915
MI 15	2586	43.2	19.7	49.9	349.6	1715	57.4	36.7	52.0	109.6	22.8	33.4	5.1	62.6	26.3	23.8	14.8	31.3	45.2	25.3	0915
ZL 16	2690	26.9	26.9	70.5	344.6	1760	86.6	23.4	64.6	81.8	70.5	21.3	8.1	110.8	14.5	25.0	22.6	21.3	29.3	13.7	0915
Mean	2936	39.0	27.0	62.6	445.7	1908	84.6	34.1	94.8	102.7	59.3	32.2	17.3	62.5	19.7	20.2	16.8	18.4	26.8	17.0	
S.E. ±	129	3.4	1.9	4.8	26.2	101	7.3	4.1	8.4	8.1	6.0	5.1	3.4	12.8	3.0	2.8	4.0	3.1	3.9	1.4	

PLASMA PHOSPHOLIPID LEVELS AFTER OPERATIONAL FLIGHTS IN THE UNITED STATES

		Total Lipid Phosphorus	Cardiolipin	Phosphatidic Acid	Phosphatidyl Glycerol	Sphingomyelin	Lecithin	Phosphatidyl Ethanolamine	Phosphatidyl Serine	Phosphatidyl Inositide	Cyclic Glycero-phosphoric Acid	Choline Plasmalogen	Ethanolamine Plasmalogen	Serine Plasmalogen	Alkyl Ethers	Inorganic Phosphorus	X_2	Alkali Labile Unknown	Unknown Plasmalogen	Acid Labile Unknowns	Cortisol MG/100 ml	Time of Sampling		
									μM Phospholipid P/liter Plasma															
IN	1	4432	66.3	52.1	52.3	883.3	2660	100.6	31.9	141.8	130.8	98.4	53.6	19.1	149.4	26.1	33.2	41.7	15.1	35.0	12.1	1930		
LE	2	3099	44.3	36.9	50.6	444.3	1665	139.7	40.3	217.3	75.9	63.2	28.2	16.4	77.3	39.3	27.3	74.7	0	24.5	10.7	1515		
MG	3	2458	24.8	21.1	29.7	395.7	1430	74.5	31.5	82.4	117.5	117.5	50.1	16.0	47.9	25.6	24.8	24.8	17.0	28.5	15.0	1100		
MO	4	2734	39.1	22.1	45.7	473.0	1605	118.4	65.3	92.7	117.0	41.3	16.7	2.7	51.9	21.3	36.9	70.0	2.7	29.0	11.9	1545		
PO	5	3562	48.1	19.2	43.8	579.5	2223	106.1	52.0	123.6	150.0	85.8	49.2	24.6	75.5	20.3	36.3	40.3	17.5	28.1	21.3	1145		
HY	6	2636	33.2	33.2	56.7	373.0	1553	107.3	23.7	104.7	158.2	63.0	43.0	19.2	54.8	31.9	34.0	27.4	9.5	27.9	11.9	1530		
HU	7	2221	30.2	17.1	30.4	347.6	1472	53.5	18.0	67.3	53.5	38.9	22.7	11.1	53.1	35.1	13.5	29.5	2.9	27.3	23.6	1200		
SM	8	3523	28.5	29.2	21.1	521.7	2563	41.6	28.5	83.1	125.4	43.7	40.9	13.4	55.3	17.3	14.4	14.4	5.3	44.0	10.6	1540		
BA	9	2744	15.1	16.2	32.1	400.1	1841	32.1	17.0	139.1	63.9	68.3	44.5	4.7	67.8	26.6	22.2	50.2	13.2	33.5	18.8	1615		
IV	10	2584	22.7	17.8	66.7	290.1	1782	61.5	28.9	92.2	100.0	43.1	21.7	4.7	48.6	23.8	36.7	26.4	21.7	23.3	11.9	1430		
MA	11	2502	23.0	14.5	45.8	284.7	1819	44.0	25.8	100.8	65.8	33.8	19.3	3.3	74.3	12.8	14.5	13.5	16.3	22.8	15.8	1600		
DI	12	2564	31.0	3.3	35.4	349.7	1802	56.7	23.6	141.8	45.5	46.9	23.3	0	61.8	19.5	16.2	11.8	15.9	22.6	17.4	1230		
MA	13	3553	45.1	33.4	45.8	504.9	2437	148.2	37.0	126.1	77.8	57.9	20.3	0	76.0	11.0	27.0	41.6	15.3	22.7	10.6	1200		
OW	14	4263	46.9	24.7	43.5	653.6	2969	162.0	67.4	180.8	155.6	56.7	21.3	9.8	50.7	29.4	17.9	14.1	11.5	25.6	10.0	1215		
ZL	15	2966	29.1	5.9	33.8	309.6	2220	105.6	37.1	89.6	91.9	28.5	25.5	9.8	40.6	13.9	32.6	19.0	5.6	18.7	10.2	1100		
Mean		3056	34.8	23.1	42.2	452.7	2017	90.1	35.2	118.9	105.2	59.1	32.0	10.3	65.7	23.6	25.8	33.3	11.3	27.6	14.1			
S.E.		173	3.1	3.2	3.1	40.4	122	10.7	4.0	10.5	10.9	6.5	3.4	2.0	6.7	2.1	2.3	5.1	1.7	1.6	1.1			

PLASMA PHOSPHOLIPIDS OF MENTALLY RETARDED CHILDREN

Subject	Total Lipid Phosphorus	Cardiolipin	Phosphatidic Acid	Phosphatidyl Glycerol	Sphingomyelin	Phosphatidyl Ethanolamine	Lecithin	Phosphatidyl Serine	Phosphatidyl Inositide	Cyclic Glycero-phosphoric Acid	Inorganic Phosphate	X_2	Alkali Labile Unknown	Choline Plasmalogen	Ethanolamine Plasmalogen	Serine Plasmalogen	Unknown Plasmalogen	Acid Labile Unknown	Alkyl Ethers	Age
	μM Phospholipid P/liter plasma																			
1	2404	18.3	0	25.7	478.4	71.9	1472	16.1	104.3	91.4	27.2	27.2	16.8	115.9	21.2	6.0	5.3	21.6	23.1	1½
2	2269	22.0	18.4	30.4	343.0	47.4	1390	17.2	131.6	61.7	21.1	22.0	27.9	111.4	32.2	6.8	31.1	26.5	44.5	
3	2992	46.4	23.9	47.6	507.2	50.3	1845	35.0	41.3	101.1	37.4	14.1	17.7	154.1	24.8	11.1	8.4	64.6	78.1	1½
4	1945	17.9	19.8	44.7	401.5	37.9	1137	14.8	86.2	44.4	7.4	27.6	19.8	71.4	6.2	17.5	1.9	26.1	51.9	9½
5	2669	29.9	19.8	32.0	542.9	47.0	1661	16.0	81.7	59.0	17.1	13.3	4.8	105.7	17.1	0	7.5	44.6	87.5	17
6	2525	39.9	32.6	56.5	397.9	52.3	1457	44.9	167.4	87.6	33.6	27.3	27.3	112.9	0	16.2	11.9	41.2	49.0	5
7	3004	16.5	12.0	33.6	459.0	68.8	2074	20.1	55.0	87.7	15.9	21.0	13.2	101.2	0	14.7	3.6	43.0	65.2	5
8	2830	24.9	9.1	44.7	463.0	60.0	1779	15.3	100.5	79.5	11.3	14.4	38.5	146.6	20.7	24.3	11.6	13.0	103.3	2½
9	2476	30.5	24.8	58.9	443.7	42.3	1356	24.8	111.4	71.6	24.8	6.2	18.6	192.6	18.6	16.8	23.8	24.3	95.3	8
10	2780	38.9	12.2	41.4	590.7	46.4	1530	13.3	87.8	91.2	8.1	19.7	15.3	147.3	40.9	17.5	22.0	39.8	129.0	15
11	2366	25.3	9.2	37.8	340.9	45.2	1520	18.9	119.0	75.0	7.1	22.7	6.9	111.9	20.1	16.8	23.9	4.3	79.0	11½
12	3300	31.4	16.5	51.5	642.3	61.7	2115	30.0	87.8	74.9	16.5	20.8	17.5	138.9	27.7	16.5	14.2	10.6	71.0	8
13	2223	29.6	16.0	46.0	279.7	40.5	1564	14.7	59.6	44.0	9.8	17.3	18.0	72.9	11.1	2.7	2.7	19.8	80.3	11½
14	2386	28.6	10.7	45.3	350.2	39.6	1571	25.5	88.5	53.4	21.7	47.0	37.0	61.1	16.0	0	0	3.6	121.7	8½
15	2927	42.1	23.4	57.7	528.0	39.8	1863	29.6	113.6	87.8	9.7	47.7	27.8	62.9	16.7	0	2.9	5.0	107.1	12
Mean	2606	29.5	16.6	43.6	451.2	50.1	1622	22.4	95.7	74.0	17.9	23.2	20.5	113.8	18.2	11.1	11.4	25.9	79.1	
Standard Deviation ±	363	9.1	8.1	10.2	101.1	10.8	269	9.1	31.6	18.0	9.6	11.4	9.6	37.6	11.1	7.9	9.7	17.8	29.6	
Standard Error ±	97	2.4	2.2	2.7	27.0	2.9	72	2.4	8.4	4.8	2.6	3.1	2.5	10.0	3.0	2.1	2.5	4.6	7.9	

PLASMA PHOSPHOLIPIDS OF CONVALESCENT CHILDREN

Subject	Total Lipid Phosphorus	Cardiolipin	Phosphatidic Acid	Phosphatidyl Glycerol	Sphingomyelin	Phosphatidyl Ethanolamine	Lecithin	Phosphatidyl Serine	Phosphatidyl Inositide	Cyclic Glycero-phosphoric Acid	Inorganic Phosphate	X_2	Alkali Labile Unknown	Choline Plasmalogen	Ethanolamine Plasmalogen	Serine Plasmalogen	Unknown Plasmalogen	Acid Labile Unknown	Alkyl Ether	Age	Original Diagnosis
	μM Phospholipid P/liter plasma																				
MC 1	2224	19.4	11.6	28.9	344.1	28.9	1533	15.8	87.8	73.3	18.2	19.4	8.4	36.3	14.7	0	3.4	21.3	39.2	15yr.	scaliosis
MM 2	2321	23.1	16.3	23.7	349.8	25.9	1567	8.7	75.5	75.8	14.2	20.9	35.1	43.0	15.1	3.6	2.7	52.7	45.3	8 yr.	coartation of the aorta
RS 3	1955	25.3	17.2	22.0	280.4	43.7	1276	16.4	40.8	52.1	13.1	20.9	0	34.3	18.3	0	4.8	28.2	54.7	10 mos.	mastoiditis
SR 4	3792	37.3	12.7	28.9	409.2	49.4	2493	21.0	81.1	85.6	19.1	29.0	9.8	50.9	15.8	14.0	6.6	32.2	118.7	14 mos.	pneumonitis
MD 5	2791	27.9	13.9	17.3	486.3	40.3	1828	24.1	93.0	97.4	11.3	29.2	11.3	41.0	16.0	11.3	8.5	25.1	47.4	20 mos.	pneumonitis
TG 6	2265	14.9	6.9	14.8	307.3	40.8	1578	16.8	77.4	72.2	25.6	22.6	24.7	37.8	10.6	9.9	2.4	17.0	58.5	9 yrs.	rheumatic fever
JL 7	2725	21.9	19.3	18.9	381.4	35.6	1746	15.0	66.2	108.3	15.0	27.6	9.7	41.2	24.7	18.0	9.1	48.4	67.7	9 yrs.	rheumatic fever
JD 8	2220	13.6	11.1	19.5	288.2	26.9	1368	22.2	67.3	70.8	16.1	19.7	5.0	27.7	8.4	11.1	5.5	55.2	55.2	11 yr.	rheumatic fever
WH 9	2212	41.6	19.9	41.3	301.0	28.6	1435	22.8	146.4	119.8	20.5	27.3	22.8	32.1	9.9	13.6	7.4	30.3	60.2	11 yr.	thyroidectomy (hyperthyroidism)
Mean	2501	25.0	14.3	23.9	349.7	35.6	1647	18.1	81.7	83.9	17.0	24.1	14.1	38.3	14.8	9.1	5.6	34.5	60.8		
S.D. ±	551	9.4	4.2	8.1	67.1	8.4	361	4.9	28.6	21.1	4.4	4.1	11.1	6.8	4.9	6.4	2.5	14.1	23.6		
S.E. ±	195	3.3	1.5	2.9	23.7	3.0	128	1.7	10.1	7.5	1.6	1.4	3.9	2.4	1.7	2.3	.9	5.0	8.3		

COMPARISON OF DIURNAL VARIATION AND ACCELERATION STRESS
ON PLASMA PHOSPHOLIPID LEVELS

Subject: UL 1

Phospholipid	First Day: Before Breakfast 8:00	First Day: After Breakfast 9:30	First Day: After Lunch 1300	First Day: End of Work Day 1530	Second Day: Acceleration Before	Second Day: Acceleration After
			µM P/liter plasma			
Total Lipid Phosphorus	2143	2527	2255	2301	2366	2346
Lecithin	1408	1775	1450	1631	1634	1466
Phosphatidyl Ethanolamine	48.4	93.5	54.6	47.6	50.2	89.6
Phosphatidyl Serine	31.3	19.5	28.2	18.9	33.8	39.7
Cardiolipin	41.4	21.0	44.0	40.7	38.1	50.4
Phosphatidic Acid	9.4	14.4	10.6	17.0	14.0	15.5
Phosphatidyl Glycerol	30.2	18.5	29.1	37.7	28.6	74.8
Phosphatidyl Inositide	111.2	48.5	84.4	102.9	114.0	151.6
Cyclic Glycerophosphoric Acid	106.7	107.2	114.8	96.2	78.6	102.8
Inorganic Phosphate	9.4	21.0	20.3	18.2	13.3	15.5
X_2	31.3	14.7	31.1	25.8	27.7	7.7
Alkali Labile Unknowns	31.3	13.7	27.5	18.2	41.6	21.8
Choline Plasmalogen	28.9	24.8	41.3	37.3	47.4	36.4
Ethanolamine Plasmalogen	14.6	15.4	22.6	15.2	25.6	22.5
Serine Plasmalogen	11.4	12.4	15.6	7.8	11.1	2.8
Unknown Plasmalogen	11.4	6.1	13.3	8.5	2.8	0
Sphingomyelin	259.9	365.7	290.5	235.7	247.3	269.8
Acid Labile Unknowns	17.1	11.4	15.3	28.1	2.8	6.1
Alkyl Ethers	52.5	58.4	68.3	36.8	85.2	99.2
Cortisol	16.0	15.5	8.0	10.5	18.3	21.8
Corticosterone	.83	.72	.76	.29	1.60	.84

COMPARISON OF DIURNAL VARIATION AND ACCELERATION STRESS
ON PLASMA PHOSPHOLIPID LEVLES

Subject: JO 2

Phospholipid	First Day: Before Breakfast 8:00	First Day: After Breakfast 9:30	First Day: After Lunch 1300	First Day: End of Work Day 1530	Second Day: Acceleration Before	Second Day: Acceleration After
	μM P/liter plasma					
Total Lipid Phosphorus	2042	2298	2087	2561	2109	1881
Lecithin	1458	1654	1249	1651	1612	1321
Phosphatidyl Ethanolamine	24.7	65.3	54.9	62.5	29.9	27.8
Phosphatidyl Serine	17.2	9.9	27.6	22.0	8.0	17.5
Cardiolipin	38.2	19.1	33.2	28.2	31.6	31.8
Phosphatidic Acid	13.1	3.9	25.3	14.1	14.1	13.7
Phosphatidyl Glycerol	18.4	21.8	40.3	43.8	22.8	48.2
Phosphatidyl Inositide	109.4	57.9	106.9	145.5	38.2	44.6
Cyclic Glycerophosphoric Acid	68.4	54.0	110.6	115.8	47.7	42.1
Inorganic Phosphate	13.1	14.0	25.3	38.7	5.7	7.7
X_2	19.6	20.2	28.0	53.0	8.7	18.3
Alkali Labile Unknowns	24.7	13.1	41.3	15.9	7.0	24.3
Choline Plasmalogen	40.2	23.2	61.2	36.4	37.5	20.1
Ethanolamine Plasmalogen	18.6	11.7	14.6	18.7	28.3	5.5
Serine Plasmalogen	8.8	13.8	13.8	14.1	11.8	0
Unknown Plasmalogen	16.3	8.7	12.5	8.5	1.3	0
Sphingomyelin	200.9	354.1	278.9	342.1	207.9	245.7
Acid Labile Unknowns	15.9	20.5	23.2	21.5	2.7	0.8
Alkyl Ethers	50.6	42.5	42.4	64.3	95.3	99.7
Cortisol	12.9	16.4	9.3	6.2	14.9	21.6
Corticosterone	.69	.54	.89	.20	.62	1.36

COMPARISON OF DIURNAL VARIATION AND ACCELERATION STRESS
ON PLASMA PHOSPHOLIPID LEVELS

Subject: WI 3

Phospholipid	Before Breakfast 8:00	After Breakfast 9:30	After Lunch 1300	End of Work Day 1530		Acceleration Before	Acceleration After
			First Day			Second Day	
			μM P/liter		plasma		
Total Lipid Phosphorus	2429	2354	2310	2668		2638	2535
Lecithin	lost	1627	1458	1711		1923	1657
Phosphatidyl Ethanolamine	lost	36.3	56.6	51.5		53.8	102.2
Phosphatidyl Serine	lost	11.8	16.6	33.9		18.2	24.3
Cardiolipin	lost	33.4	28.2	52.3		41.7	55.3
Phosphatidic Acid	lost	14.8	20.8	25.6		26.1	20.0
Phosphatidyl Glycerol	lost	25.0	30.5	31.5		24.5	49.2
Phosphatidyl Inositide	lost	85.9	109.7	125.7		96.3	56.8
Cyclic Glycerophosphoric Acid	lost	64.5	124.0	142.2		56.5	82.4
Inorganic Phosphate	lost	8.5	15.7	18.4		12.1	26.6
X_2	lost	17.4	31.0	23.2		4.5	3.6
Alkali Labile Unknowns	lost	11.8	36.7	30.2		26.7	17.5
Choline Plasmalogen	lost	34.4	52.4	37.9		39.0	52.2
Ethanolamine Plasmalogen	lost	14.6	14.8	25.1		19.5	23.8
Serine Plasmalogen	lost	7.1	10.4	13.3		4.0	7.1
Unknown Plasmalogen	lost	6.4	3.9	8.0		3.2	2.0
Sphingomyelin	lost	337.8	320.4	346.6		315.3	341.7
Acid Labile Unknowns	lost	9.4	28.0	22.4		10.6	10.4
Alkyl Ethers	lost	124.5	66.8	103.3		99.2	112.8
Cortisol	14.2	18.6	14.4	7.7		10.1	9.6
Corticosterone	.48	.79	.72	.31		.19	.12

COMPARISON OF DIURNAL VARIATION AND ACCELERATION STRESS
ON PLASMA PHOSPHOLIPID LEVELS

Subject: WIN 4

Phospholipic	First Day: Before Breakfast 8:00	First Day: After Breakfast 9:30	First Day: After Lunch 1300	First Day: End of Work Day 1530	Second Day: Acceleration Before	Second Day: Acceleration After
	μM P/liter plasma					
Total Lipid Phosphorus	2078	2106	1811	2518	1945	1989
Lecithin	1319	1493	1143	1553	1354	1317
Phosphatidyl Ethanolamine	31.6	55.4	28.6	56.4	25.1	33.6
Phosphatidyl Serine	12.1	17.1	21.4	39.8	16.1	18.5
Cardiolipin	36.2	23.2	23.4	44.6	16.7	25.9
Phosphatidic Acid	15.4	20.6	15.8	15.1	19.5	15.5
Phosphatidyl Glycerol	20.4	34.1	28.6	35.8	14.0	29.2
Phosphatidyl Inositide	93.1	65.3	102.7	158.4	61.3	59.3
Cyclic Glycerophosphoric Acid	98.1	88.3	102.3	116.3	54.5	63.5
Inorganic Phosphate	10.2	22.1	16.1	18.1	19.5	8.6
X_2	25.8	5.9	32.2	16.6	17.5	11.1
Alkali Labile Unknowns	23.1	11.0	20.5	34.2	16.9	8.4
Choline Plasmalogen	32.2	20.2	36.8	36.3	33.8	43.2
Ethanolamine Plasmalogen	15.0	2.5	9.4	15.4	9.9	9.8
Serine Plasmalogen	10.8	8.2	7.6	11.8	3.3	5.4
Unknown Plasmalogen	19.9	4.4	3.8	7.8	2.7	0
Sphingomyelin	339.9	284.1	255.7	404.6	293.1	344.3
Acid Labile Unknowns	22.7	20.0	22.8	34.0	0	0
Alkyl Ethers	53.4	33.9	33.9	52.9	81.7	87.5
Cortisol	13.2	25.6	9.7	10.5	10.7	19.4
Corticosterone	.59	1.08	.08	.67	.24	.29

COMPARISON OF DIURNAL VARIATION AND ACCELERATION STRESS
ON PLASMA PHOSPHOLIPID LEVELS

Subject: HA 5

Phospholipid	First Day: Before Breakfast 8:00	First Day: After Breakfast 9:30	First Day: After Lunch 1300	First Day: End of Work Day 1530	Second Day: Acceleration Before	Second Day: Acceleration After
	μM P/liter Plasma					
Total Lipid Phosphorus	1696	2099	1704	1880	1768	1768
Lecithin	1061	1437	1082	1108	1053	1081
Phosphatidyl Ethanolamine	48.0	73.7	36.3	59.4	58.7	65.1
Phosphatidyl Serine	24.8	26.0	9.4	28.4	25.5	10.4
Cardiolipin	34.9	28.8	22.2	32.7	30.9	23.2
Phosphatidic Acid	8.8	12.6	13.3	15.8	10.8	17.3
Phosphatidyl Glycerol	21.0	33.0	31.2	23.7	19.6	45.8
Phosphatidyl Inositide	59.9	88.2	143.3	72.6	66.6	48.8
Cyclic Glycerophosphoric Acid	85.1	63.6	89.3	75.4	74.1	42.1
Inorganic Phosphate	13.6	18.5	9.4	18.1	17.0	23.5
X_2	24.3	13.6	19.1	35.0	20.9	14.0
Alkali Labile Unknowns	20.2	16.6	29.0	20.1	20.0	31.8
Choline Plasmalogen	23.1	47.2	31.9	34.2	29.9	37.1
Ethanolamine Plasmalogen	17.8	14.9	12.8	22.0	15.7	16.1
Serine Plasmalogen	10.2	12.2	11.8	13.4	6.7	8.8
Unknown Plasmalogen	15.1	8.4	5.8	21.3	10.8	3.0
Sphingomyelin	255.2	239.3	208.1	301.6	306.2	296.4
Acid Labile Unknowns	14.3	15.7	21.8	36.5	24.2	21.4
Alkyl Ethers	35.3	56.7	30.0	45.7	56.0	56.7
Cortisol	9.9	19.2	22.1	23.5	10.9	12.2
Corticosterone	.40	1.36	1.69	1.50	.28	.79

COMPARISON OF DIURNAL VARIATION AND ACCELERATION STRESS
ON PLASMA PHOSPHOLIPID LEVLES

Subject: RO 6

Phospholipid	First Day: Before Breakfast 8:00	First Day: After Breakfast 9:30	First Day: After Lunch 1300	First Day: End of Work Day 1530	Second Day: Acceleration Before	Second Day: Acceleration After
	μM P/liter plasma					
Total Lipid Phosphorus	1777	1980	1767	1767	1556	1707
Lecithin	1015	1279	1142	1102	1031	1148
Phosphatidyl Ethanolamine	34.3	45.3	39.0	48.4	19.5	44.2
Phosphatidyl Serine	10.3	15.8	18.7	22.1	9.8	10.1
Cardiolipin	30.6	25.0	22.8	17.0	20.9	20.0
Phosphatidic Acid	13.0	7.9	11.0	11.8	12.0	24.8
Phosphatidyl Glycerol	39.1	18.6	26.3	29.5	21.3	32.1
Phosphatidyl Inositide	154.0	71.9	71.6	87.1	143.3	39.6
Cyclic Glycerophosphoric Acid	132.0	77.6	107.4	76.7	28.6	58.4
Inorganic Phosphate	18.3	18.6	8.7	21.2	7.2	3.4
X_2	26.7	8.3	27.9	26.2	8.7	5.0
Alkali Labile Unknowns	35.0	10.3	22.8	18.9	17.9	27.3
Choline Plasmalogen	38.0	31.5	27.4	30.9	28.0	30.6
Ethanolamine Plasmalogen	11.2	18.6	13.3	12.2	4.7	26.6
Serine Plasmalogen	10.5	13.7	11.5	13.8	4.7	12.6
Unknown Plasmalogen	17.4	6.1	13.3	7.6	0	7.5
Sphingomyelin	251.6	355.2	231.6	266.5	239.5	226.0
Acid Labile Unknowns	12.1	10.9	11.5	10.6	0	1.2
Alkyl Ethers	29.5	39.4	45.2	52.0	58.7	66.2
Cortisol	12.0	13.8	10.3	12.0	10.9	17.7
Corticosterone	.67	.61	.40	.44	.35	1.31

COMPARISON OF DIURNAL VARIATION AND ACCELERATION STRESS
ON PLASMA PHOSPHOLIPID LEVELS

Subject: YO 7

Phospholipid	First Day: Before Breakfast 8:00	First Day: After Breakfast 9:30	First Day: After Lunch 1300	First Day: End of Work Day 1530		Second Day: Acceleration Before	Second Day: Acceleration After
			μM P/liter plasma				
Total Lipid Phosphorus	2816	2905	2626	3220		2839	2405
Lecithin	1750	1992	1728	2160		2045	1613
Phosphatidyl Ethanolamine	65.1	62.5	50.9	71.5		51.4	42.1
Phosphatidyl Serine	27.6	16.9	13.7	43.5		20.7	22.1
Cardiolipin	39.7	48.8	32.0	42.2		29.8	38.5
Phosphatidic Acid	17.2	5.8	12.9	38.6		17.3	21.9
Phosphatidyl Glycerol	42.5	13.4	42.0	49.6		19.9	41.1
Phosphatidyl Inositide	116.9	109.5	86.9	162.6		81.2	110.9
Cyclic Glycerophosphoric Acid	151.0	90.9	96.1	97.9		68.4	85.4
Inorganic Phosphate	24.5	12.5	19.2	14.2		9.7	17.6
X_2	36.9	14.2	21.0	17.7		11.9	29.6
Alkali Labile Unknowns	34.6	9.9	42.3	34.1		22.2	20.7
Choline Plasmalogen	34.6	24.4	63.0	60.9		48.0	39.4
Ethanolamine Plasmalogen	23.7	14.2	17.3	24.8		10.8	9.1
Serine Plasmalogen	16.3	18.9	13.7	10.0		6.8	4.3
Unknown Plasmalogen	18.0	8.4	13.7	8.4		4.5	5.8
Sphingomyelin	478.5	526.1	404.1	455.0		372.2	307.3
Acid Labile Unknowns	14.1	28.8	18.4	15.5		16.2	11.3
Alkyl Ethers	57.7	52.0	73.0	83.1		144.0	107.5
Cortisol	13.1	10.4	13.3	8.0		10.5	7.6
Corticosterone	.54	.41	.34	.55		.64	.76

COMPARISON OF DIURNAL VARIATION AND ACCELERATION STRESS ON PLASMA PHOSPHOLIPID LEVELS

Subject: KI 8

Phospholipid	First Day: Before Breakfast 8:00	First Day: After Breakfast 9:30	First Day: After Lunch 1300	First Day: End of Work Day 1530	Second Day: Acceleration Before	Second Day: Acceleration After
			μM P/liter plasma			
Total Lipid Phosphorus	2061	2109	2330	2362	1962	2044
Lecithin	1281	1325	1267	1430	1371	1233
Phosphatidyl Ethanolamine	34.6	54.2	46.4	59.0	18.2	72.1
Phosphatidyl Serine	22.7	19.8	34.5	31.9	20.4	24.1
Cardiolipin	35.9	27.2	59.4	40.2	36.5	22.1
Phosphatidic Acid	18.5	17.1	14.7	11.3	13.5	18.4
Phosphatidyl Glycerol	31.9	25.1	41.5	28.1	15.7	36.6
Phosphatidyl Inositide	99.7	90.5	219.7	102.0	72.8	89.1
Cyclic Glycerophosphoric Acid	104.3	83.3	178.3	127.3	38.8	94.4
Inorganic Phosphate	24.5	19.8	17.2	14.6	16.9	15.7
X_2	40.8	9.3	28.9	35.7	11.8	21.3
Alkali Labile Unknowns	35.9	12.9	43.8	31.9	10.0	23.1
Choline Plasmalogen	28.0	42.2	35.4	45.6	33.4	37.6
Ethanolamine Plasmalogen	14.2	23.8	22.8	25.7	12.6	9.4
Serine Plasmalogen	14.8	17.7	18.2	8.5	8.8	5.9
Unknown Plasmalogen	19.6	9.7	15.6	5.0	3.1	0
Sphingomyelin	286.0	289.7	327.6	349.5	273.2	329.4
Acid Labile Unknowns	9.5	42.8	24.5	38.3	15.7	15.1
Alkyl Ethers	59.8	99.3	69.7	87.2	87.1	91.6
Cortisol	15.2	11.0	17.8	18.6	21.0	21.2
Corticosterone	.57	.35	.75	1.32	1.80	1.70

COMPARISON OF DIURNAL VARIATION AND ACCELERATION STRESS
ON PLASMA PHOSPHOLIPID LEVELS

Subject: BL 9

	First Day					Second Day	
	Before Breakfast	After Breakfast	After Lunch	End of Work Day		Acceleration	Acceleration
	8:00	9:30	1300	1530		Before	After
Phospholipid			μM P/liter plasma				
Total Lipid Phosphorus	1939	1918	2310	2290		1768	2081
Lecithin	1298	1300	1503	1376		1237	1394
Phosphatidyl Ethanolamine	45.8	40.7	74.2	48.1		28.6	61.4
Phosphatidyl Serine	33.4	10.0	30.3	28.4		13.6	14.4
Cardiolipin	35.3	16.9	47.6	33.7		24.9	17.1
Phosphatidic Acid	10.1	5.0	33.7	25.0		19.6	14.4
Phosphatidyl Glycerol	30.6	19.9	43.4	35.5		13.4	34.6
Phosphatidyl Inositide	68.1	58.9	131.4	111.8		64.2	87.0
Cyclic Glycerophosphoric Acid	89.4	61.8	87.8	129.2		45.1	63.5
Inorganic Phosphate	17.7	9.2	14.1	21.8		12.0	10.8
X_2	13.0	10.0	38.1	24.1		12.0	22.1
Alkali Labile Unknowns	19.4	13.4	30.3	15.1		8.1	20.4
Choline Plasmalogen	29.7	25.3	31.4	38.5		22.1	41.6
Ethanolamine Plasmalogen	17.1	10.6	18.7	20.8		17.7	12.9
Serine Plasmalogen	9.7	12.7	15.5	14.0		7.4	8.7
Unknown Plasmalogen	0	7.7	9.7	8.0		5.3	9.4
Sphingomyelin	267.3	318.5	252.3	369.7		246.9	292.0
Acid Labile Unknowns	8.2	50.2	28.4	38.9		17.7	14.8
Alkyl Ethers	40.3	37.8	41.8	62.1		60.5	66.0
Cortisol	15.9	18.5	19.9	8.1		16.0	15.0
Corticosterone	.81	.65	1.02	.21		.36	.43

COMPARISON OF DIURNAL VARIATION AND ACCELERATION STRESS
ON PLASMA PHOSPHOLIPID LEVELS

Subject: BR 10

	First Day					Second Day	
	Before Breakfast	After Breakfast	After Lunch	End of Work Day		Acceleration	Acceleration
	8:00	9:30	1300	1530		Before	After
Phospholipid			μM P/liter plasma				
Total Lipid Phosphorus	2005	2587	2440	2847		1998	2118
Lecithin	1292	1855	1579	1761		1401	1552
Phosphatidyl Ethanolamine	27.1	51.5	42.7	61.2		24.6	38.3
Phosphatidyl Serine	16.4	16.8	28.1	45.3		21.6	15.3
Cardiolipin	32.3	27.4	43.9	46.4		22.4	21.8
Phosphatidic Acid	14.0	13.7	14.6	19.7		10.4	11.2
Phosphatidyl Glycerol	31.5	32.9	24.9	51.3		18.8	29.7
Phosphatidyl Inositide	97.7	119.5	144.9	126.7		56.3	47.2
Cyclic Glycerophosphoric Acid	76.6	81.2	96.9	140.4		48.4	43.8
Inorganic Phosphate	24.5	15.5	22.7	16.8		9.4	5.1
X_2	21.1	7.2	37.1	41.6		6.0	5.1
Alkali Labile Unknowns	32.3	6.5	22.5	39.0		5.4	8.7
Choline Plasmalogen	29.9	37.8	43.4	43.6		41.8	39.2
Ethanolamine Plasmalogen	14.6	16.0	21.2	14.8		17.6	22.0
Serine Plasmalogen	13.2	14.8	11.5	10.8		11.4	9.3
Unknown Plasmalogen	11.8	6.2	12.4	14.8		10.6	10.8
Sphingomyelin	321.5	307.9	348.4	453.3		313.5	298.4
Acid Labile Unknowns	12.6	45.5	16.8	22.8		17.2	8.1
Alkyl Ethers	37.5	70.9	59.8	73.2		56.9	53.4
Cortisol	15.1	16.1	17.2	8.6		11.0	19.2
Corticosterone	.40	.64	.66	.35		.46	1.51

COMPARISON OF DIURNAL VARIATION AND ACCELERATION STRESS
ON PLASMA PHOSPHOLIPID LEVELS

Subject: MA 11

Phospholipid	First Day: Before Breakfast 8:00	First Day: After Breakfast 9:30	First Day: After Lunch 1300	First Day: End of Work Day 1530		Second Day: Acceleration Before	Second Day: Acceleration After
			μM P/liter plasma				
Total Lipid Phosphorus	2051	2395	2574	2732		1639	2268
Lecithin	1267	1579	1750	1881		1084	1543
Phosphatidyl Ethanolamine	45.1	61.1	66.7	45.9		26.4	34.7
Phosphatidyl Serine	28.7	20.1	25.7	26.0		18.4	16.3
Cardiolipin	30.2	28.5	43.5	38.2		31.5	29.0
Phosphatidic Acid	12.1	20.6	25.7	16.1		15.1	15.2
Phosphatidyl Glycerol	36.7	29.5	36.0	32.2		19.5	39.7
Phosphatidyl Inositide	99.1	136.5	122.8	115.8		90.3	90.1
Cyclic Glycerophosphoric Acid	118.6	125.3	101.4	69.7		49.8	83.3
Inorganic Phosphate	13.1	16.8	27.3	30.1		14.8	12.3
X_2	30.4	14.6	23.7	29.0		9.3	22.5
Alkali Labile Unknowns	35.5	12.2	30.4	35.0		17.4	20.9
Choline Plasmalogen	35.9	37.1	45.8	33.9		31.6	39.0
Ethanolamine Plasmalogen	18.1	18.0	20.6	20.2		19.5	18.8
Serine Plasmalogen	15.8	9.8	19.0	17.8		7.7	5.9
Unknown Plasmalogen	8.4	6.5	17.5	7.1		9.2	9.3
Sphingomyelin	264.0	326.5	254.0	378.6		205.0	331.6
Acid Labile Unknowns	29.7	28.0	30.4	25.4		15.6	14.7
Alkyl Ethers	62.6	53.4	67.9	69.4		60.8	54.7
Cortisol	12.0	17.1	12.0	11.1		12.1	17.4
Corticosterone	.31	.36	.26	.46		.41	.75

COMPARISON OF DIURNAL VARIATION AND ACCELERATION STRESS
ON PLASMA PHOSPHOLIPID LEVELS

Subject: FR 12

Phospholipid	First Day: Before Breakfast 8:00	First Day: After Breakfast 9:30	First Day: After Lunch 1300	First Day: End of Work Day 1530		Second Day: Acceleration Before	Second Day: Acceleration After
			µM P/liter plasma				
Total Lipid Phosphorus	2742	3039	-	-		2990	2625
Lecithin	1862	2112	-	-		2314	1883
Phosphatidyl Ethanolamine	61.4	55.0	-	-		52.9	54.3
Phosphatidyl Serine	23.6	25.2	-	-		20.9	20.5
Cardiolipin	41.4	33.4	-	-		27.5	20.7
Phosphatidic Acid	15.6	17.3	-	-		9.0	11.0
Phosphatidyl Glycerol	35.9	50.5	-	-		42.5	54.3
Phosphatidyl Inositide	110.8	152.0	-	-		83.4	112.9
Cyclic Glycerophosphoric Acid	145.1	117.9	-	-		92.7	90.8
Inorganic Phosphate	16.2	20.7	-	-		6.6	18.4
X_2	33.5	12.8	-	-		15.9	26.3
Alkali Labile Unknowns	23.3	23.1	-	-		12.6	20.7
Choline Plasmalogen	42.8	47.1	-	-		41.3	51.2
Ethanolamine Plasmalogen	21.9	13.7	-	-		19.7	23.6
Serine Plasmalogen	18.7	15.8	-	-		14.1	14.7
Unknown Plasmalogen	7.1	6.7	-	-		11.7	12.9
Sphingomyelin	361.2	371.4	-	-		321.1	300.1
Acid Labile Unknowns	21.9	41.3	-	-		10.8	11.6
Alkyl Ethers	36.8	86.6	-	-		48.1	37.3
Cortisol	12.0	10.4	-	-		12.4	9.7
Corticosterone	.26	.05	-	-		.39	.51

COMPARISON OF DIURNAL VARIATION AND ACCELERATION STRESS
ON PLASMA PHOSPHOLIPID LEVLES

Subject: WIL 13

Phospholipid		First Day: Before Breakfast 8:00	First Day: After Breakfast 9:30	First Day: After Lunch 1300	First Day: End of Work Day 1500		Second Day: Acceleration Before	Second Day: Acceleration After
		μM P/liter plasma						
Total Lipid Phosphorus	2691							
Lecithin	1988							
Phosphatidyl Ethanolamine	64.3							
Phosphatidyl Serine	21.5							
Cardiolipin	26.9							
Phosphatidic Acid	14.3							
Phosphatidyl Glycerol	23.7							
Phosphatidyl Inositide	82.9							
Cyclic Glycerophosphoric Acid	116.3							
Inorganic Phosphate	7.3							
X_2	15.6							
Alkali Labile Unknowns	27.5							
Choline Plasmalogen	39.0							
Ethanolamine Plasmalogen	10.5							
Serine Plasmalogen	15.1							
Unknown Plasmalogen	2.2							
Sphingomyelin	315.1							
Acid Labile Unknowns	7.8							
Alkyl Ethers	49.5							
Cortisol	13.4							
Corticosterone	.44							

COMPARISON OF DIURNAL VARIATION AND ACCELERATION STRESS
ON PLASMA PHOSPHOLIPID LEVELS

Subject: JA 14

Phospholipid	First Day: Before Breakfast 8:00	First Day: After Breakfast 9:30	First Day: After Lunch 1300	First Day: End of Work Day 1530	Second Day: Acceleration Before	Second Day: Acceleration After
			µM P/liter plasma			
Total Lipid Phosphorus	2179	2231	2285	2763	1903	2429
Lecithin	1331	1516	1485	1752	1255	1666
Phosphatidyl Ethanolamine	64.5	49.1	47.3	58.8	53.7	67.5
Phosphatidyl Serine	25.5	15.2	25.8	39.8	21.3	32.8
Cardiolipin	39.0	20.5	36.8	46.1	12.5	20.2
Phosphatidic Acid	13.5	13.6	12.6	24.3	8.0	28.7
Phosphatidyl Glycerol	24.8	28.8	25.8	52.8	41.1	57.8
Phosphatidyl Inositide	82.1	77.4	101.0	111.6	78.2	121.0
Cyclic Glycerophosphoric Acid	132.7	71.4	104.4	133.7	95.0	127.0
Inorganic Phosphate	24.8	8.3	13.3	22.1	5.1	12.6
X_2	27.2	16.7	16.9	22.1	19.4	22.1
Alkali Labile Unknowns	19.8	12.1	25.1	24.3	28.6	24.5
Choline Plasmalogen	45.5	31.0	32.5	54.2	44.9	42.8
Ethanolamine Plasmalogen	20.7	23.0	20.8	23.5	24.0	18.5
Serine Plasmalogen	14.2	14.7	10.5	17.7	17.1	13.4
Unknown Plasmalogen	16.8	6.3	20.1	8.3	12.9	10.0
Sphingomyelin	332.3	333.8	364.9	434.3	224.6	230.8
Acid Labile Unknowns	20.7	37.0	26.5	31.8	15.0	16.3
Alkyl Ethers	42.9	64.0	29.3	35.6	20.6	46.9
Cortisol	14.1	12.4	10.1	8.2	10.2	8.5
Corticosterone	.81	.70	.72	.62	.58	.25

COMPARISON OF DIURNAL VARIATION AND ACCELERATION STRESS
ON PLASMA PHOSPHOLIPID LEVELS

Subject: EV 15

Phospholipid	First Day: Before Breakfast 8:00	First Day: After Breakfast 9:30	First Day: After Lunch 1300	First Day: End of Work Day 1530	Second Day: Acceleration Before	Second Day: Acceleration After
			μM P/liter plasma			
Total Lipid Phosphorus	2009	1855	1630	1931	2198	2120
Lecithin	1403	1194	1071	1231	1394	1430
Phosphatidyl Ethanolamine	28.3	40.3	36.0	51.0	50.8	57.2
Phosphatidyl Serine	13.3	19.7	21.4	22.0	20.2	15.1
Cardiolipin	23.3	27.5	32.1	43.5	43.5	23.5
Phosphatidic Acid	12.3	8.9	13.2	18.0	16.7	21.2
Phosphatidyl Glycerol	32.4	20.8	30.0	34.6	38.7	75.5
Phosphatidyl Inositide	72.5	91.3	88.5	91.2	182.6	82.0
Cyclic Glycerophosphoric Acid	109.5	76.8	58.5	67.0	133.8	68.0
Inorganic Phosphate	3.0	10.4	14.0	11.2	26.2	9.8
X_2	12.1	15.0	20.5	27.8	10.8	15.3
Alkali Labile Unknowns	10.1	9.8	19.7	11.2	17.8	12.7
Choline Plasmalogen	39.6	31.9	25.8	47.3	56.3	54.9
Ethanolamine Plasmalogen	16.1	26.3	15.5	11.2	23.7	27.6
Serine Plasmalogen	11.1	9.8	8.2	10.8	18.0	20.1
Unknown Plasmalogen	0	7.8	13.7	7.7	10.6	18.0
Sphingomyelin	267.2	238.7	203.3	274.4	216.7	230.4
Acid Labile Unknowns	7.6	42.7	10.9	30.1	15.4	11.0
Alkyl Ethers	48.4	76.8	33.9	37.9	53.0	51.7
Cortisol	14.3	14.1	13.9	9.8	10.8	11.5
Corticosterone	.51	.78	.59	.35	.82	.83

COMPARISON OF DIURNAL VARIATION AND ACCELERATION STRESS
ON PLASMA PHOSPHOLIPID LEVELS

Subject: DP 16

Phospholipid	First Day: Before Breakfast 8:00	First Day: After Breakfast 9:30	First Day: After Lunch 1300	First Day: End of Work Day 1530		Second Day: Acceleration Before	Second Day: Acceleration After
	μM P/liter plasma						
Total Lipid Phosphorus	2160	2235	2402	2701		2356	1929
Lecithin	1493	1439	1598	1618		1682	1169
Phosphatidyl Ethanolamine	45.6	45.8	72.5	73.5		42.9	58.4
Phosphatidyl Serine	14.5	29.5	34.4	23.0		16.3	21.6
Cardiolipin	37.2	31.7	44.4	37.3		22.9	39.0
Phosphatidic Acid	11.0	13.6	30.3	15.7		8.3	33.4
Phosphatidyl Glycerol	18.6	21.7	35.6	40.8		36.5	68.5
Phosphatidyl Inositide	75.6	98.1	94.4	151.0		114.1	124.8
Cyclic Glycerophosphoric Acid	94.6	91.7	86.5	148.8		92.8	105.1
Inorganic Phosphate	12.5	18.3	13.9	23.5		9.0	8.3
X_2	22.5	34.7	25.9	33.2		16.3	30.1
Alkali Labile Unknowns	21.8	47.4	33.2	37.8		24.3	15.8
Choline Plasmalogen	50.5	19.0	42.0	53.2		53.7	52.5
Ethanolamine Plasmalogen	19.9	8.7	16.6	24.6		24.7	10.0
Serine Plasmalogen	7.8	13.6	14.7	8.6		18.4	6.8
Unknown Plasmalogen	8.6	2.7	3.8	7.0		14.9	1.7
Sphingomyelin	276.9	343.3	286.3	445.1		232.3	242.2
Acid Labile Unknowns	9.9	49.9	21.4	45.4		18.4	14.1
Alkyl Ethers	44.9	36.2	66.5	48.6		56.8	28.9
Cortisol	9.5	14.7	15.3	9.2		11.7	12.1
Corticosterone	.32	.79	.54	.35		.40	.39

COMPARISON OF DIURNAL VARIATION AND ACCELERATION STRESS
ON PLASMA PHOSPHOLIPID LEVELS

Subject: WID 17

	First Day					Second Day	
	Before Breakfast	After Breakfast	After Lunch	End of Work Day		Acceleration	Acceleration
	8:00	9:30	1300	1530		Before	After
Phospholipid			μM P/liter plasma				
Total Lipid Phosphorus	1878	1846	2144	2071		1824	2000
Lecithin	1247	1150	1457	1321		1237	1287
Phosphatidyl Ethanolamine	36.4	67.6	60.7	67.5		37.8	44.2
Phosphatidyl Serine	11.8	26.0	29.4	21.8		15.0	11.2
Cardiolipin	19.5	31.6	33.9	40.8		20.8	19.4
Phosphatidic Acid	8.8	18.1	15.9	11.6		12.0	10.4
Phosphatidyl Glycerol	23.5	28.3	18.9	35.4		40.5	53.2
Phosphatidyl Inositide	89.0	101.2	96.3	126.1		93.9	92.6
Cyclic Glycerophosphoric Acid	97.3	101.4	109.6	93.8		82.1	121.0
Inorganic Phosphate	11.5	7.0	14.2	13.3		6.4	19.0
X_2	17.3	24.7	17.8	34.6		19.9	16.8
Alkali Labile Unknowns	16.5	15.9	33.9	33.8		22.3	2.8
Choline Plasmalogen	34.0	40.8	34.9	41.6		39.6	44.4
Ethanolamine Plasmalogen	17.1	14.4	17.4	11.6		15.5	22.8
Serine Plasmalogen	14.1	13.3	11.2	7.9		19.7	9.0
Unknown Plasmalogen	3.6	10.0	9.2	5.8		6.9	6.6
Sphingomyelin	266.8	219.7	243.8	248.6		151.4	261.6
Acid Labile Unknowns	10.3	20.5	18.9	18.2		25.7	16.4
Alkyl Ethers	48.6	49.9	30.7	48.9		74.6	60.8
Cortisol	15.0	12.5	14.2	7.5		11.6	15.6
Corticosterone	.67	.39	.56	.29		.43	.49

COMPARISON OF DIURNAL VARIATION AND ACCELERATION STRESS
ON PLASMA PHOSPHOLIPID LEVELS

Subject: PA 18

	First Day					Second Day	
	Before Breakfast	After Breakfast	After Lunch	End of Work Day		Acceleration	Acceleration
	8:00	9:30	1300	1530		Before	After
Phospholipid			μM P/liter plasma				
Total Lipid Phosphorus	2035	1904	2036	2179		2109	1764
Lecithin	1356	1317	1272	1355		1364	1097
Phosphatidyl Ethanolamine	48.6	53.5	47.0	65.6		40.5	37.2
Phosphatidyl Serine	34.0	22.7	19.1	25.5		17.9	12.7
Cardiolipin	34.6	18.7	33.4	29.9		32.9	15.7
Phosphatidic Acid	18.1	7.8	14.9	20.0		15.6	26.3
Phosphatidyl Glycerol	38.5	26.1	31.8	36.6		32.7	54.2
Phosphatidyl Inositide	88.9	69.1	108.7	93.3		104.2	70.6
Cyclic Glycerophosphoric Acid	93.0	83.6	102.8	109.6		98.1	81.1
Inorganic Phosphate	9.4	18.7	13.6	13.7		8.0	4.1
X_2	19.3	20.4	20.8	21.1		15.6	16.2
Alkali Labile Unknowns	26.1	13.1	28.9	18.3		20.2	9.0
Choline Plasmalogen	36.2	21.0	37.3	37.5		62.6	44.6
Ethanolamine Plasmalogen	13.6	11.1	27.3	18.7		17.5	19.4
Serine Plasmalogen	8.3	2.7	6.3	15.5		6.5	13.2
Unknown Plasmalogen	7.7	11.1	18.1	10.2		4.0	15.2
Sphingomyelin	236.2	242.3	307.3	331.0		304.7	268.3
Acid Labile Unknowns	12.6	24.0	22.8	30.5		16.7	21.5
Alkyl Ethers	55.4	36.0	27.3	50.8		53.6	39.7
Cortisol	16.3	12.4	9.0	6.9		12.4	12.0
Corticosterone	.61	.29	.07	.08		.28	.52

COMPARISON OF DIURNAL VARIATION AND ACCELERATION STRESS ON PLASMA PHOSPHOLIPID LEVELS

Subject: OL 19

	First Day					Second Day	
	Before Breakfast	After Breakfast	After Lunch	End of Work Day		Acceleration	Acceleration
	8:00	9:30	1300	1530		Before	After
Phospholipid			μM P/liter plasma				
Total Lipid Phosphorus	-	-	2902	2784		2588	2752
Lecithin	-	-	1886	1719		1772	1911
Phosphatidyl Ethanolamine	-	-	59.2	49.8		53.3	59.2
Phosphatidyl Serine	-	-	32.5	28.3		27.4	22.6
Cardiolipin	-	-	35.1	36.5		33.6	25.1
Phosphatidic Acid	-	-	14.5	lost		10.9	23.7
Phosphatidyl Glycerol	-	-	34.8	26.9		21.5	56.7
Phosphatidyl Inositide	-	-	181.1	lost		82.3	54.0
Cyclic Glycerophosphoric Acid	-	-	125.1	lost		106.6	70.5
Inorganic Phosphate	-	-	22.1	lost		20.2	19.3
X_2	-	-	27.9	lost		20.2	25.1
Alkali Labile Unknowns	-	-	31.6	lost		27.4	16.8
Choline Plasmalogen	-	-	48.2	50.1		44.5	44.9
Ethanolamine Plasmalogen	-	-	31.6	27.1		16.3	22.6
Serine Plasmalogen	-	-	6.7	19.8		6.5	19.3
Unknown Plasmalogen	-	-	15.4	14.7		13.7	18.7
Sphingomyelin	-	-	433.0	480.6		390.2	417.8
Acid Labile Unknowns	-	-	18.3	28.2		17.6	16.8
Alkyl Ethers	-	-	58.6	34.0		47.9	51.8
Cortisol			17.0	9.8		23.1	12.1
Corticosterone			.61	.46		1.05	.38

COMPARISON OF DIURNAL VARIATION AND ACCELERATION STRESS
ON PLASMA PHOSPHOLIPID LEVELS

Subject: MH 20

Phospholipid	First Day: Before Breakfast 8:00	First Day: After Breakfast 9:30	First Day: After Lunch 1300	First Day: End of Work Day 1530	Second Day: Acceleration Before	Second Day: Acceleration After
			μM P/liter plasma			
Total Lipid Phosphorus	-	-	-	-	1824	1981
Lecithin	-	-	-	-	1144	1292
Phosphatidyl Ethanolamine	-	-	-	-	30.3	39.4
Phosphatidyl Serine	-	-	-	-	11.3	18.8
Cardiolipin	-	-	-	-	21.5	14.7
Phosphatidic Acid	-	-	-	-	12.8	17.2
Phosphatidyl Glycerol	-	-	-	-	30.3	44.7
Phosphatidyl Inositide	-	-	-	-	114.4	100.2
Cyclic Glycerophosphoric Acid	-	-	-	-	96.8	57.8
Inorganic Phosphate	-	-	-	-	17.5	16.8
X_2	-	-	-	-	22.3	18.8
Alkali Labile Unknowns	-	-	-	-	22.3	19.6
Choline Plasmalogen	-	-	-	-	42.0	37.8
Ethanolamine Plasmalogen	-	-	-	-	26.8	14.1
Serine Plasmalogen	-	-	-	-	13.0	19.6
Unknown Plasmalogen	-	-	-	-	18.1	11.1
Sphingomyelin	-	-	-	-	246.0	292.0
Acid Labile Unknowns	-	-	-	-	16.6	19.6
Alkyl Ethers	-	-	-	-	35.9	47.7
Cortisol	-	-	-	-	14.7	21.8
Corticosterone	-	-	-	-	.50	2.03

COMPARISON OF DIURNAL VARIATION AND ACCELERATION STRESS
ON PLASMA PHOSPHOLIPID LEVLES

Subject: JON 21

	First Day					Second Day	
	Before Breakfast	After Breakfast	After Lunch	End of Work Day		Acceleration	Acceleration
	8:00	9:30	1300	1530		Before	After
Phospholipid			μM P/liter plasma				
Total Lipid Phosphorus	-	-	-	-		2019	2190
Lecithin	-	-	-	-		1410	1271
Phosphatidyl Ethanolamine	-	-	-	-		28.5	67.0
Phosphatidyl Serine	-	-	-	-		14.9	24.1
Cardiolipin	-	-	-	-		18.4	21.5
Phosphatidic Acid	-	-	-	-		11.5	22.3
Phosphatidyl Glycerol	--	-	-	-		27.3	63.1
Phosphatidyl Inositide	-	-	-	-		60.0	135.1
Cyclic Glycerophosphoric Acid	-	-	-	-		85.0	115.0
Inorganic Phosphate	-	-	-	-		7.5	16.4
X_2	-	-	-	-		14.9	28.5
Alkali Labile Unknowns	-	-	-	-		20.0	25.4
Choline Plasmalogen	-	-	-	-		42.6	45.8
Ethanolamine Plasmalogen	-	-	-	-		11.7	22.8
Serine Plasmalogen	-	-	-	-		13.5	12.9
Unknown Plasmalogen	-	-	-	-		6.3	9.2
Sphingomyelin	-	-	-	-		269.5	325.0
Acid Labile Unknowns	-	-	-	-		20.0	19.9
Alkyl Ethers	-	-	-	-		54.3	75.3
Cortisol	-	-	-	-		7.4	8.0
Corticosterone	-	-	-	-		.41	.45

END

END

UNCLASSIFIED

Security Classification

DOCUMENT CONTROL DATA - R & D

(Security classification of title, body of abstract and indexing annotation must be entered when the overall report is classified)

1. ORIGINATING ACTIVITY *(Corporate author)*
Aerospace Medical Research Department
U.S. Naval Air Development Center
Johnsville, Warminster, Pa. 18974

2a. REPORT SECURITY CLASSIFICATION
Unclassified

2b. GROUP

3. REPORT TITLE
Effect Of Physical And Psychic Stress On Phosphatidyl Glycerol And Related Phospholipids

4. DESCRIPTIVE NOTES *(Type of report and inclusive dates)*
Phase Report

5. AUTHOR(S) *(First name, middle initial, last name)*
B. David Polis, Edith Polis, John deCani, H. P. Schwarz, Lorraine Dreisbach

6. REPORT DATE	7a. TOTAL NO. OF PAGES	7b. NO. OF REFS
	27	21

8a. CONTRACT OR GRANT NO.
Air Task A34 531 062/2021/F022 01 06

b. PROJECT NO. (5314-062-59)
Air Task R01 101 01 (RC-3-01)

c.
Task MR005.04.0098, Task MF12.524.005-7006

d.

9a. ORIGINATOR'S REPORT NUMBER(S)
NADC-MR-6805

9b. OTHER REPORT NO(S) *(Any other numbers that may be assigned this report)*

10. DISTRIBUTION STATEMENT
This document is subject to special export controls and each transmittal to foreign governments or foreign nationals may be made only with prior approval of Naval Air Development Center, Johnsville, Warminster, Pa. 18974.

11. SUPPLEMENTARY NOTES

12. SPONSORING MILITARY ACTIVITY

13. ABSTRACT Chromatographic analyses of phospholipids in tissue and plasma of rats exposed to lethal levels of ionizing radiation or acceleration stress yielded a consistent pattern of increased concentrations of phosphatidyl glycerol. Extension of the studies to humans stressed by acceleration to grayout, sleep deprivation, schizophrenia, combat, etc., revealed that all stresses were accompanied by significant increments in plasma phosphatidyl glycerol. Moreover, the stressed populations could be distinguished from each other when the changes in phosphatidyl glycerol were related to concomitant variations in seven other phospholipids. Using a statistical method involving discriminant function analysis, a function Z was obtained which represented the summation of the log of each individual phospholipid concentration times a distributive constant. With this analysis the phospholipid distribution separated normal combat, and schizophrenic population into three distinct groups. In animal experiments it was found that hypophysectomy, which markedly enhanced the tolerance of the rat to acceleration stress, abolished the plasma changes of acceleration and caused a two-fold increase in the brain level of phosphatidyl glycerol after exposure to acceleration. The results from human and animal experiments are interpreted to indicate that some center of the brain can interpret certain sensory inputs as threats to survival and reacts by mobilizing biochemical factors at a molecular level to meet this threat and enhance survival. In all the stresses studied, with the exception of schizophrenia, the individuals returned to normal levels with rest. The maintenance of this "locked stress" pattern in the schizophrenic and the reversible plasma phospholipid changes in the other physical and psychic stresses in human coupled with the tissue phospholipid changes in rat offer an approach to the cerebral control factors operative in the biochemistry of environmental and pathological stress.

DD FORM 1 NOV 65 1473 (PAGE 1)

UNCLASSIFIED

Security Classification

14. KEY WORDS	LINK A		LINK B		LINK C	
	ROLE	WT	ROLE	WT	ROLE	WT
1. Phospholipids						
2. Phosphatidyl Glycerol						
3. Stress						
4. Biochemical Mechanisms						
5. Acceleration						
6. Schizophrenia						
7. Combat Stress						

Naval Air Development Center,
Johnsville, Warminster, Pa.
Aerospace Medical Research Department
EFFECT OF PHYSICAL AND PSYCHIC STRESS ON PHOSPHATIDYL GLYCEROL AND RELATED PHOSPHOLIPIDS, by B. David Polis, Edith Polis, John deCani, H.P. Schwarz, Lorraine Dreisbach.
1 August 1968. 27 p.

UNCLASSIFIED
NOFORN

Includes references.

1. Phospholipids
2. Phosphatidyl Glycerol
3. Stress
4. Biochemical Mechanisms
I. B.David Polis
II. NADC-MR-6805

AirTask A34 531
062/2021/F022 01
06 (5314-062-59)
UNCLASSIFIED

Naval Air Development Center,
Johnsville, Warminster, Pa.
Aerospace Medical Research Department
EFFECT OF PHYSICAL AND PSYCHIC STRESS ON PHOSPHATIDYL GLYCEROL AND RELATED PHOSPHOLIPIDS, by B. David Polis, Edith Polis, John deCani, H.P. Schwarz, Lorraine Dreisbach.
1 August 1968. 27 p.

UNCLASSIFIED
NOFORN

Includes references.

1. Phospholipids
2. Phosphatidyl Glycerol
3. Stress
4. Biochemical Mechanisms
I. B.David Polis
II. NADC-MR-6805

AirTask A34 531
062/2021/F022 01
06 (5314-062-59)
UNCLASSIFIED

Naval Air Development Center
Johnsville, Warminster, Pa.
Aerospace Medical Research Department
EFFECT OF PHYSICAL AND PSYCHIC STRESS ON PHOSPHATIDYL GLYCEROL AND RELATED PHOSPHOLIPIDS, by B. David Polis, Edith Polis, John deCani, H.P. Schwarz, Lorraine Dreisbach.
1 August 1968. 27 p.

UNCLASSIFIED
NOFORN

Includes references.

1. Phospholipids
2. Phosphatidyl Glycerol
3. Stress
4. Biochemical Mechanisms
I. B.David Polis
II. NADC-MR-6805

AirTask A34 531
062/2021/F022 01
06 (5314-062-59)
UNCLASSIFIED

Naval Air Development Center,
Johnsville, Warminster, Pa.
Aerospace Medical Research Department
EFFECT OF PHYSICAL AND PSYCHIC STRESS ON PHOSPHATIDYL GLYCEROL AND RELATED PHOSPHOLIPIDS, by B. David Polis, Edith Polis, John deCani, H.P. Schwarz, Lorraine Dreisbach.
1 August 1968. 27 p.

UNCLASSIFIED
NOFORN

Includes references.

1. Phospholipids
2. Phosphatidyl Glycerol
3. Stress
4. Biochemical Mechanisms
I. B.David Polis
II. NADC-MR-6805

AirTask A34 531
062/2021/F022 01
06 (5314-062-59)
UNCLASSIFIED

Naval Air Development Center,
Johnsville, Warminster, Pa.
Aerospace Medical Research Department
EFFECT OF PHYSICAL AND PSYCHIC STRESS ON PHOSPHATIDYL GLYCEROL AND RELATED PHOSPHOLIPIDS, by B. David Polis, Edith Polis, John deCani, H.P. Schwarz, Lorraine Dreisbach.
1 August 1968. 27 p.

UNCLASSIFIED
NOFORN

Includes references.

1. Phospholipids
2. Phosphatidyl Glycerol
3. Stress
4. Biochemical Mechanisms

I. B.David Polis
II. NADC-MR-6805

AirTask A34 531
062/2021/F022 01
06 (5314-062-59)
UNCLASSIFIED

Naval Air Development Center,
Johnsville, Warminster, Pa.
Aerospace Medical Research Department
EFFECT OF PHYSICAL AND PSYCHIC STRESS ON PHOSPHATIDYL GLYCEROL AND RELATED PHOSPHOLIPIDS, by B. David Polis, Edith Polis, John deCani, H.P. Schwarz, Lorraine Dreisbach.
1 August 1968. 27 p.

UNCLASSIFIED
NOFORN

Includes references.

1. Phospholipids
2. Phosphatidyl Glycerol
3. Stress
4. Biochemical Mechanisms

I. B.David Polis
II. NADC-MR-6805

AirTask A34 531
062/2021/F022 01
06 (5314-062-59)
UNCLASSIFIED

Naval Air Development Center
Johnsville, Warminster, Pa.
Aerospace Medical Research Department
EFFECT OF PHYSICAL AND PSYCHIC STRESS ON PHOSPHATIDYL GLYCEROL AND RELATED PHOSPHOLIPIDS, by B. David Polis, Edith Polis, John deCani, H.P. Schwarz, Lorraine Dreisbach.
1 August 1968. 27 p.

UNCLASSIFIED
NOFORN

Includes references.

1. Phospholipids
2. Phosphatidyl Glycerol
3. Stress
4. Biochemical Mechanisms

I. B.David Polis
II. NADC-MR-6805

AirTask A34 531
062/2021/F022 01
06 (5314-062-59)
UNCLASSIFIED

Naval Air Development Center,
Johnsville, Warminster, Pa.
Aerospace Medical Research Department
EFFECT OF PHYSICAL AND PSYCHIC STRESS ON PHOSPHATIDYL GLYCEROL AND RELATED PHOSPHOLIPIDS, by B. David Polis, Edith Polis, John deCani, H.P. Schwarz, Lorraine Dreisbach.
1 August 1968. 27 p.

UNCLASSIFIED
NOFORN

Includes references.

1. Phospholipids
2. Phosphatidyl Glycerol
3. Stress
4. Biochemical Mechanisms

I. B.David Polis
II. NADC-MR-6805

AirTask A34 531
062/2021/F022 01
06 (5314-062-59)
UNCLASSIFIED

DISTRIBUTION LIST

DDC, Alexandria, Virginia ---------- 20
National Library of Medicine, Bethesda, Md. ----------
Bureau of Medicine and Surgery (Code 71) ----------
Bureau of Medicine and Surgery (Code 522) ---------- 3
Naval Aerospace Medical Institute, Pensacola, Fla. ---------- 1
NMRI (Tech. Ref. Library), Bethesda, Md. ---------- 1
USN Submarine Medical Center (Library), New London, Conn. ----------
Naval Air Systems Command (AIR-531) ---------- 1
Naval Air Systems Command (AIR-604) ---------- 2
Naval Air Systems Command (AIR-5311G) ----------
USN Medical Neuropsychiatric Research Unit, San Diego, Calif. ----------
Naval Safety Center, Norfolk, Va. ----------
Naval Air Test Center, Patuxent River, Md. ----------
ONR (Code 107), Washington, D.C. ----------
ONR, FPO New York, N.Y. ----------
FAA (AC-100), Oklahoma City, Okla. ----------
FAA (Medical Library, HQ-630), Washington, D.C. ----------
FAA (Aviation Medical Div.), Kansas City, Mo. ----------
FAA (AC-922), Oklahoma City, Okla. ----------
[illegible] AMRL (MRBB), Wright-Patterson AFB, Ohio ----------
[illegible] AMRL (MR[illegible]), Wright-Patterson AFB, Ohio ----------
[illegible] USAF [illegible], Washington, D.C. ----------
[illegible] School of Aerospace Med. [illegible], Brooks AFB, Tex. ----------
[illegible] SAM (SMBP[illegible]), Brooks AFB, Tex. ----------
[illegible] SAM (SMKOR), Brooks AFB, Tex. ----------
[illegible] University Library, Maxwell AFB, Ala. ----------
[illegible] Flight Test Center [illegible], Edwards AFB, Calif. ----------
[illegible] TAC [illegible], Langley AFB, [illegible] ----------
[illegible] Pathology Branch, Washington, D.C. ----------
[illegible] Edgewood Arsenal, Md. ----------
[illegible] Natick Laboratories [illegible], Natick, Mass. ----------
[illegible] Research Inst. of Environmental Med. [illegible]CE), Natick, Mass. ----------
[illegible] Research Laboratory, Fort Knox, Ky. ----------
NASA Lewis Research Center [illegible], Cleveland, Ohio ----------
NASA Manned Spacecraft Center [illegible], Houston, Tex. ----------
NASA Scientific & Tech. Information Fac. [illegible]DL), College Park, Md. ----------
Library of Congress [illegible], Washington, D.C. ----------
[illegible] Institutes of Health [illegible], Bethesda, Md. ----------
[illegible] Research Council [illegible], Washington, D.C. ----------
[illegible] Science Board, Nat. Academy of Sciences (NRC), Washington [illegible]

[illegible] Foundation for Research [illegible] ----------
[illegible] Medicine Research [illegible], Columbus, Ohio ----------
[illegible] Abstracts, Philadelphia, Pa. ----------
[illegible] State College (Psychology Dept.), California, Pa. ----------
[illegible] Aeronautical Laboratory [illegible], Buffalo, N.Y. ----------
[illegible] Guggenheim Aviation Safety [illegible], [illegible]ne, N.J. ----------
Countway Library of Medicine, Boston, Mass. ----------
Drexel Institute of Technology (Biomedical Engrg.), Phila., Pa.

DISTRIBUTION LIST (Contld)

University of Illinois, Chicago, Ill.
Indiana University (Library), Indianapolis, Ind.
Library of College of Physicians of Philadelphia, Phila., Pa.
Lovelace Foundation, Albuquerque, N.M.
Ohio State University, Columbus, Ohio
University of Pennsylvania (Physiology Dept.), Philadelphia, Pa.
John B. Pierce Foundation (Dr J.D. Hardy), New Haven, Conn.
Presbyterian-Univ. of Pa. Medical Center (B.F. Burgess), Phila., Pa.
Princeton University, Princeton, N.J.
University of Southern California, Los Angeles, Calif.
Temple University Hospital (Dr. Kern), Philadelphia, Pa.
University of Washington (D. Monteith), Seattle, Wash.
Wayne State University (Medical Library), Detroit, Mich.
Wrightsville Marine Bio-Medical Laboratory, Wilmington, N.C.